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DENTAL PERSONNEL

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ANY discussion of dentosocial problems, whether they be on a national, state or local level, must, of necessity, take into consideration dental personnel. No one can seriously question the statement that public appreciation of good dental health has increased tremendously during the past twenty-five years. Few will question the prediction that the public's appreciation of good dental health will continue to increase at an even more accelerated rate in the future. Popular appreciation is the forerunner of popular demand, and popular demand in dentistry, as in all other walks of life, must be met by adequate supply.

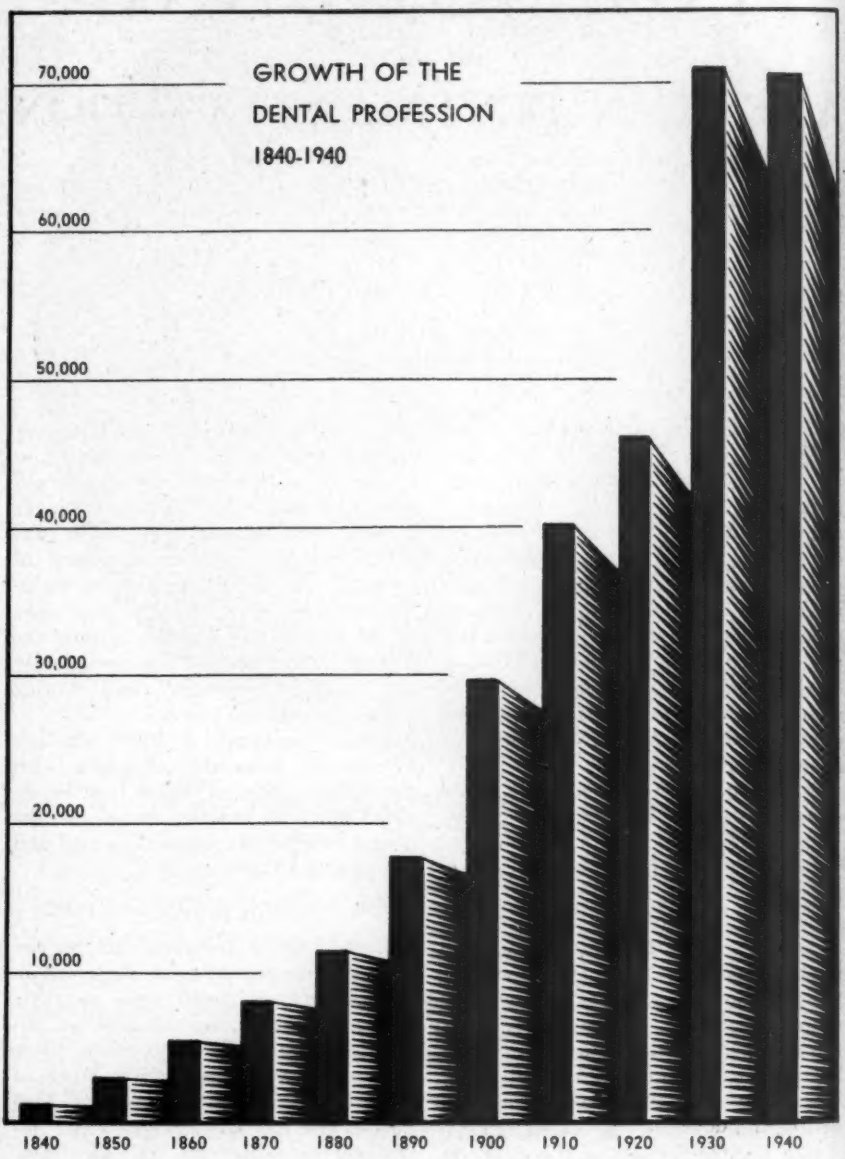
The American Dental Association is fully aware of the public's growing appreciation of mouth health and of the growing demand for dental service. It is apprehensive over the fact that the profession may not, under present conditions, or under conditions that are likely to exist in the immediate future, be able to meet those demands with the quality of service that the public desires and deserves.

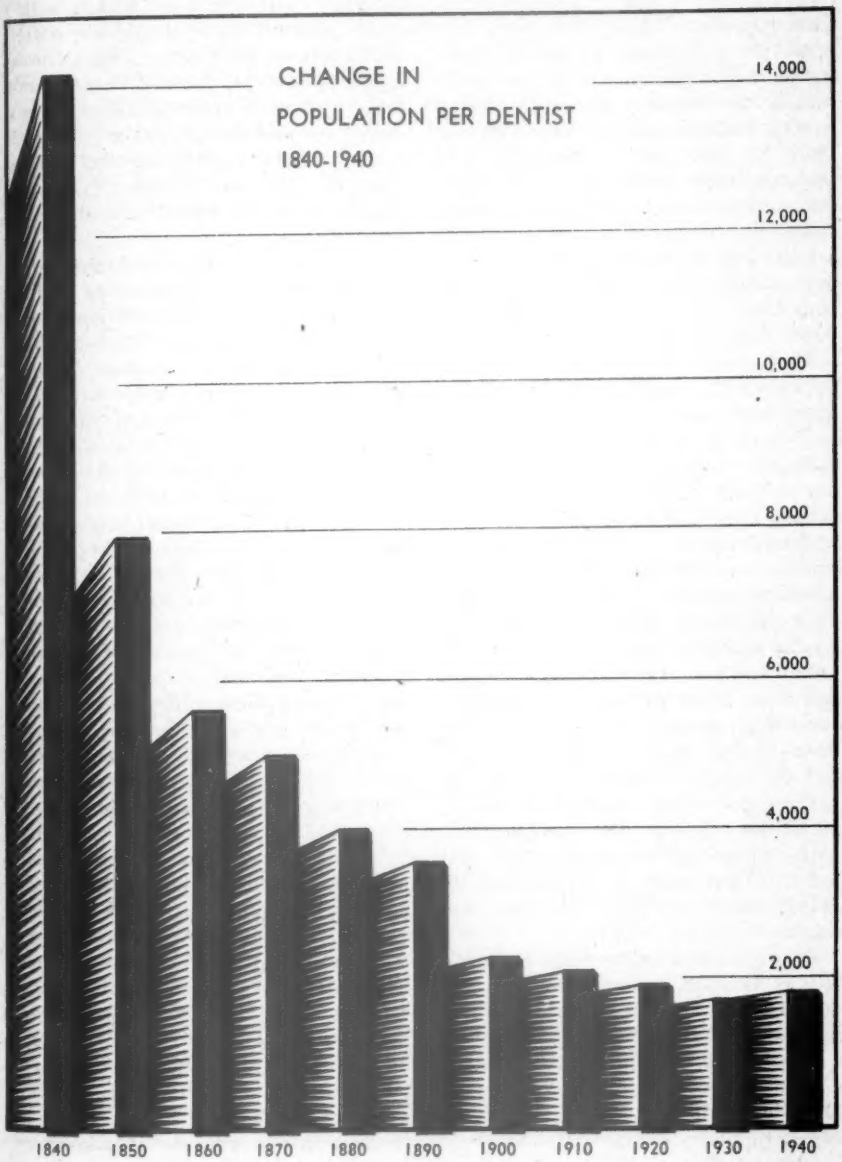
It is not the purpose of this paper to offer any new or startling device or method that might be employed to satisfy public demand. Rather, it will attempt to review past and present trends in both dental demand and dental supply in the hope that a comparison of yesterday with today may help to evaluate tomorrow, and that it may assist in the preparation of plans to meet tomorrow's conditions. "I know no way of judging the future," said Patrick Henry, "but by the past."

Dental personnel, or those who are directly or indirectly concerned with the service of dental supply, can be divided into four groups: the dentist, the dental assistant, the dental hygienist and the technician.

THE DENTIST

Census figures regarding dentists are not available for periods prior to 1850. It has been estimated that, in 1825, there were only 250 dentists in the United States.[1] By 1840, there were approximately 1,200 dentists, most of whom were located east of the Alleghenies in the larger cities. The remainder were itinerant craftsmen trav-


GROWTH OF THE
DENTAL PROFESSION
1840-1940
70,000
60,000
50,000
40,000
30,000
20,000
10,000
1840
1850
1860
1870
1880
1890
1900
1910
1920
1930
1940


CHANGE IN
POPULATION PER DENTIST
1840-1940
14,000
12,000
10,000
8,000
6,000
4,000
2,000
1840
1850
1860
1870
1880
1890
1900
1910
1920
1930
1940

eling from village to village. The federal census of 1850 lists a population of a little more than 23,000,000, including 2,923 dentists; a ratio of one dentist to 8,000 people. During the next fifty years, the population tripled itself, rising to almost 76,000,000. In the same period, the dental profession increased tenfold, expanding from 2,923 to 29,665. Thus, by 1900, the ratio of dentist to population was one to 2,561+. By 1910, the population had grown to approximately 92,000,000, while the number of dentists had increased one-third, to 39,997, making the ratio of dentist to population one to 2,300+. By 1920, there were 56,152 dentists serving 106,000,000 people, a ratio of one to 1,882+. By 1930, the population of the United States had increased to almost 123,000,000, while the number of dentists had increased to 71,055, making a ratio of one to 1,728. According to the sixteenth federal census, the population in 1940 was 131,669,000, and the number of dentists was listed at 70,601, making a dentist:population ratio of one to 1,865. For ordinary purposes, this figure is usually quoted as one to 1,800.

Any one who questions the statement that the trend in dental demand is other than upward need only to look at these figures. Supply follows demand, and the supply of dentists as evidenced by the decreasing ratio of dentist to population during the past hundred years is conclusive evidence that the trend in the public's appreciation of dental health has been decidedly upward.

During its first eighty years as a profession, dentistry's development in the technical and biologic aspects was little short of phenomenal. This development was responsible to a great extent for the growth in public demand, which, in turn, was reflected in the growth of dental supply.

During the past ten years, the numerical growth of the profession has remained practically static, despite the fact that appreciable advances have been made in its biologic and technical branches. Some there are who have become alarmed over dentistry's decelerated growth since 1930. This alarm is unfounded as the decrease in the number of graduates entering the profession during the past decade can be attributed to two natural conditions: the depression, the primary condition, and the lengthening of the dental curriculum, the secondary.

From 1920 to 1924, the number of graduates increased from a low of 906 to a high of 3,422. The addition of one year predental training to the curriculum resulted in a slight temporary decrease in graduates as the number of dental students graduating between 1925 and 1929 averaged about 2,500 a year. The shock of the depression in the fall of 1929 was reflected in 1930, as during that year only 1,561 students graduated in dentistry. The effect of the depression was felt throughout the entire period of the '30's, the number of dental graduates averaging a little less than 1,800 a year. The raising of the predental requirements in 1936 and 1937 was reflected temporarily in the number of 1941 graduates, the dental schools in that year graduating but 1,568 students. In 1942, the number of graduates had increased to 1,784; in 1943, to 1,926, and, in 1944, to 2,470. It should be pointed out too that the 1941 freshman class was the largest in several years, consisting of 2,476 students.

Thus, as economic conditions began to improve, students interested in dentistry began to increase; and there is every evidence that, had the war not interfered, dental enrollments would have continued to increase, despite the fact that the dental curriculum has been raised to six years. Thus, it is well to repeat that the reduction in number of dental graduates during the past fifteen years has been caused primarily by the

economic conditions of the '30's, and that the lengthening of the dental curriculum was but a secondary causal condition, having but a temporary effect.

While on the subject of dental graduates, it might be well to point out that although resulting in a temporary decrease in the quantity of dental graduates, the increase in predental training raised the quality of the students. In 1944, 49 per cent of the graduates had more than a minimum predental training, and 29 per cent had a degree before entering dentistry; which augurs well for both the public and the profession.

There is no disputing the fact that, during the past fifteen years, the supply of dentists has not kept abreast of the public appreciation and demand for service; and that, as a result, in the United States, dentistry is seriously undermanned. Any dental program, whether inaugurated on a national, state or local level, must take that fact into serious consideration.

There are many reasons that dental demands have increased so precipitously over the past fifty years, among which external stimuli such as increased per capita income, urbanization and improved transportation play a part. The principal reasons, however, are of an internal nature, consisting of the advances that dentistry has made in the biologic and technical fields and in the profession's efforts to educate the public in matters of dental health.

Diffusion among the population of a knowledge of the character and quality of modern dentistry has been the concomitant of dentistry's progress. Interestingly enough, this diffusion has taken place largely by word of mouth. The increased demand has not been created by advertising, because what little dental advertising has been offered the public has been very low in quality and effectiveness. The increased demand, therefore, has not been due to abnormal stimulation, but rather to a gradual awakening of the public to a better appreciation of dental health, brought about by professional education.

The public's appreciation of dental service, which, as we have seen, has had a gradual growth during the past fifty or more years, has suddenly taken a tremendous upswing as a result of the war effort. More than 10,000,000 men are experiencing, many for the first time, the benefits of a healthy mouth. Recognition of these benefits by a large number of service men will continue into the postwar period. It will be transformed into desires and into demands for better dental care, both for themselves and for their families. Therefore, there is every reason to expect that, for the next decade at least, the demands for dental service will materially exceed the supply.

As yet, we have discussed only the relationship of supply to dental demand. No mention has been made of dental need. In spite of the fact that dentistry has a valuable service to render to virtually every member of the community, only a small percentage of the population as yet actually purchase that service. In 1940, there was approximately one dentist to every 1,800 of the population. Yet it is estimated that, in 1929, the average dentist treated only slightly more than 400 patients a year.[2]

If this estimate is correct, the present personnel of approximately 70,000 dentists can furnish annual dental service to about 28,000,000 to 30,000,000 people, or to approximately from one-fifth to one-quarter of the population.

There are, of course, many factors other than supply and demand to be considered in discussing the rendition of dental service. One of these factors, and a very important one, is distribution. For instance, while the dentist-population ratio in 1940 was approximately one to 1,800 for the country as a whole, the distribution of dentists was by no means uniform. O'Rourke's study[3] reveals that,

in 1940, the dentist-population ratio in the New England states was 1:1,674; in the Middle Atlantic states, 1:1,457; East North Central, 1:1,630; West North Central, 1:1,591; South Atlantic, 1:3,001; East South Central, 1:4,780; West South Central, 1:3,320; Mountain, 1:2,171, and Pacific Coast states, 1:1,290.

A breakdown by geographic districts is not sufficient. Wide variations in the dentist-population ratio exists in each district. For instance, according to a survey of dentists in private practice made in December 1942 and September 1943 in thirty-two counties in Michigan,[4] four counties had no resident dentists, seven counties had one dentist per 3,000-3,500 population, three counties had one dentist per 3,500-4,000 population, four counties had one dentist per 4,500-5,000 population, three counties had one dentist per 6,000-6,500 population and three counties had one dentist per 6,500-9,500 population. This condition is not confined to Michigan. It exists to a greater or less degree in other states. For example,[5] thirty-six of Georgia's 159 counties have no resident dentist. One of Indiana's ninety-two counties has no dentist; two of the 105 counties in Kansas have no dentist; one of Missouri's 114 countries has no dentist, and thirty-four of the 254 counties in Texas have no dentists. Time does not permit a tabulation of dentists by counties in all states, but the data presented indicate a problem that must be solved before dental care can be made

POPULATION, DENTISTS AND RATIO OF POPULATION TO DENTISTS IN THE UNITED STATES, 1840 TO 1940

Year	Population	Number of Dentists	Population per Dentist
1840	17,069,453	1,200	14,224
1850	23,191,876	2,923	7,934
1860	31,443,321	5,606	5,609
1870	39,818,449	7,988	4,985
1880	50,155,783	12,314	4,073
1890	62,947,714	17,498	3,597
1900	75,994,575	29,665	2,562
1910	91,972,266	39,997	2,299
1920	105,710,620	56,152	1,883
1930	122,775,046	71,055	1,728
1940	131,669,275	70,601	1,865

Source: Comparative Occupation Statistics for the United States, 1870-1940, and other publications, U. S. Bureau of the Census, Washington, 1943.

available to many who now desire it. This problem is magnified infinitely if dental care is to be supplied to all who are in need.

O'Rourke writes[3]:

> The apparent lack of correlation between dentist : population ratio and the population per square mile is doubtless brought about by many conditions. However, it is probable that the most significant influences are the geographic, agricultural or industrial characteristics of the various regions. . . . Mountainous area, desert, farms of large acreage cause unevenness in the population distribution. Therefore, it is essential that analysis of states and geographic districts for the purpose of determining the accessibility of their populations to dental personnel include consideration of population distribution. Dentists will not tend to locate in remote or sparsely settled areas, and yet the total number of people living in such areas is considerable. Better roads and improved methods of transportation have widened the area which a dentist may serve effectively, and this condition is very likely to continue. Therefore, it is expected that increased accessibility to sources of dental service may in the future increase the demands upon dental personnel by many persons who reside in thinly populated areas.

While it is possible to anticipate with some degree of certainty an increase in demand for dental services in the post-war period, it is extremely difficult to predict what, if any, increase will take place in dental personnel to meet those demands. Any estimate regarding the number of dental graduates in the immediate future must be highly speculative. The war and Selective Service rulings will influence, if not wholly control, the number of dental enrollees and dental graduates.

In all probability, the number of graduates entering the profession during the next two years will little more

Numerical

Year	Laboratories	Dentists	Ratio
1890	2	17,498	1:8749
1930	100	56,152	1:562
1944	2,800	50,000*	1:17

Volume of Sales

1920	$ 15,000,000
1930	46,000,000
1943	100,000,000
1944	125,000,000

*Civilian dentists.

than balance the number of dentists lost by death and retirement. It is estimated that the number of dental graduates in 1945 will be approximately 2,270, while the estimated number for 1946 has been placed at 2,152. The combined mortality and retirement rate among active dentists has been conservatively estimated at twenty to twenty-four per thousand.[5] Thus, the active dentist: population ratio of approximately 1:1,800 that existed in 1940 will remain essentially the same in 1946. In view of the present unsettled conditions in educational circles, any estimate of the dentist: population ratio beyond 1946 would be merely speculative. However, judging by existing figures and by the upward trend in public appreciation of dental service, it seems safe to predict that the dental profession will be barely able to meet current dental demands and that, under present methods of practice, the profession will be exceedingly overtaxed to supply anticipated future demands.

A study of dental progress over the past eighty years shows certain significant trends, which must be clearly understood in considering the present and future dental health problem. The trend in public appreciation of dental service is upward and at an accelerated rate. It will continue upward, and even more rapidly, in the future. The trend in dental supply is also upward. However, the increase in supply of the past fifteen years has not paralleled the rise in public appreciation. On the other hand, that supply has appreciably improved in quality.

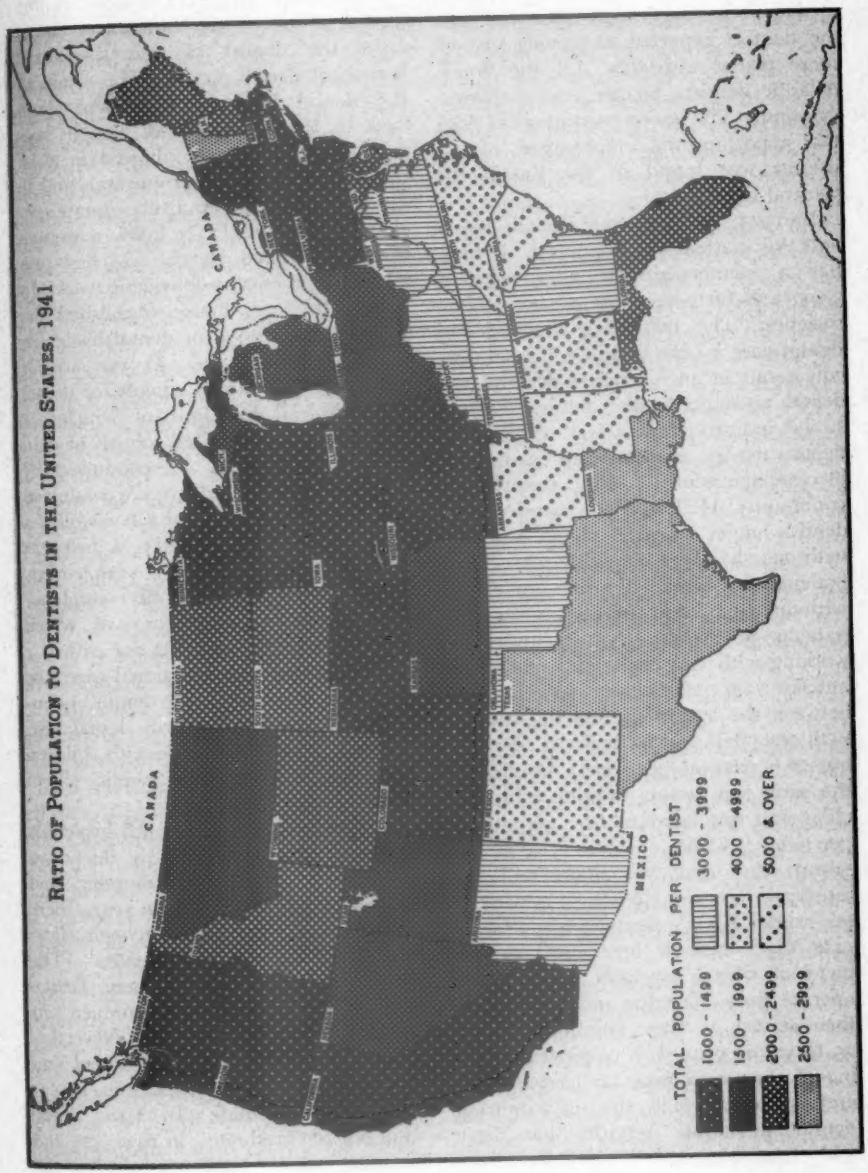
Although the ratio of dentists to population has remained static for the past fifteen or more years, the amount of dental service rendered the public has definitely increased. This has been made possible by improvements in technic and by the employment of auxiliary personnel. Auxiliary service, specifically that furnished by the assistant, the hygienist and the technician, plays an important and ever increasing part in dental supply.

THE DENTAL ASSISTANT

Early dental literature makes little reference to the use of women as dental assistants. Prior to 1850, what little assisting was done was rendered by the dental apprentice. With the passing of the apprentice system, other provisions had to be made for nontechnical aid. An article written in 1884 indicates that, at that date, women were just beginning to enter the dental assistant field.[6] As the demand for dental service increased, and as the character of dental service improved, dentists found the services of assistants increasingly indispensable, with the result that there has been a tremendous growth in this type of personnel. The utilization of the dental assistant's services has enabled the dentist to partially meet the increased demand for dental care.

Since most dentists prefer to train their assistants themselves, there has been little demand for specialized education in this field. At the present time, three schools offer a one-year course for dental assistants: Northwestern University, North Pacific College of Dentistry and Los Angeles Junior College. In addition, the U. S. Office of Education lists the Essex County Vocational High School in Newark, N.J., and the Edison Vocational High School in Mt. Vernon, N.Y., as offering courses for dental assistants.

Since dental assistants are not licensed or registered, there is no information regarding the number who enter or leave the field each year. According to the American Dental Association report, "Survey of Dental Practice," 41.3 per cent of the dentists in 1929 employed full-time assistants or nurses. These figures were confirmed by a survey made by the Committee on Economics of the


RATIO OF POPULATION TO DENTISTS IN THE UNITED STATES, 1941
CANADA
CANADA
MEXICO
TOTAL POPULATION PER DENTIST
1000 - 1499
1500 - 1999
2000 - 2499
2500 - 2999
3000 - 3999
4000 - 4999
5000 & OVER

American Dental Association in 1938. According to a study of 3,000 dentists by Klein[8] in 1943, more than one-half the dentists reported employing one or more dental assistants. Of the South Atlantic dentists, 69 per cent employed assistants. The lowest percentage of dentists reporting the employment of assistants was found in the East-North Central area, 49.1.

In 1924, the American Dental Assistants Association was organized. It now has a membership of approximately 3,000 and forty-one state or component societies. The increasing demand for dental care by the public will undoubtedly result in an increasing demand for dental assistants by the dentist. Klein's study[9] indicates that a well-trained, efficient dental assistant can materially increase the dentist's ability to serve his community. He found, for instance, that dentists under 45 years of age working with one chair and no assistant saw 47.6 patients per week. Dentists under 45 with one chair and one assistant saw 61.8 patients per week. Dentists under 45 working with two chairs and one assistant saw 72.9 patients per week. Dentists between the age of 55 and 64 working with one chair and no assistant averaged but 26.6 patients per week. Dentists in the same age group working with one chair and one assistant saw 44 patients per week; while dentists in this age group who used two chairs and one assistant were able to see 55.5 patients per week. Klein's figures seem to indicate that, through intelligent employment of dental assistants, dentists can operate more efficiently and can increase their service to their community from 25 to 50 per cent, with considerable saving of their own time and nervous energy. The trend in the utilization of dental assistants' services has been and continues to be definitely upward, and there is every indication that this trend will be accelerated in the future.

THE DENTAL HYGIENIST

The dental hygienist constitutes the second group whose function it is to assist the dentist in one specialized branch of dental service. The origin of the dental hygienist movement dates back to 1902 when C. M. Wright suggested that women be allowed to learn the technic of dental prophylaxis and to develop this field into a subordinate specialty of dentistry.[10] In 1906, a woman without a D.D.S. degree was first permitted to practice prophylactic work. In 1913, Alfred C. Fones established the first training school for dental hygienists in Bridgeport, Conn. At the present time, there are fifteen schools for dental hygienists, all but four of which are conducted by recognized schools of dentistry. Two courses are conducted by dental infirmaries and two are conducted by teacher colleges. Four schools offer a one-year course; six offer a two-year course; one offers a four-year course, and the remaining four offer combinations of one, two and four years, which will prepare the students for either a prophylactic or an educational career or both. While originally the dental hygienist was trained to perform dental prophylaxis only, her aptitude with children soon opened a field in public school work.

At the present time, dental hygienists are licensed to practice in thirty-two states, the District of Columbia and Hawaii. For the past fifteen years, there have been at least sixteen schools offering courses for dental hygienists. (The Murry and Leonie Guggenheim Dental Clinic has recently discontinued its school for dental hygienists.) Nevertheless, the enrollment in each school and the total number of graduates each year are extremely small. In 1920, there were but 123 graduates; in 1930, 373 and in 1943, 293.[11] Graduates during the intervening years were on approximately the same level.

The exact number of dental hygien-

ists practicing today is unknown. According to the *Journal of the American Dental Hygienists' Association* of October 1943, the annual number of admissions to practice does not exceed the number that retire annually. There were 3,029 registered hygienists ten years ago, with little or no increase to date. However, according to an article just received by THE JOURNAL from Miss Frances A. Stoll of Columbia University, there are approximately 7,000 hygienists now practicing in the United States. The wide variation in these two reports indicates that more complete information on this subject is needed.

At the present time, there are 1,700 members of the American Dental Hygienists' Association, which was organized in 1927. The association has twenty-six state component societies.

Klein's[8] recently published survey of 1,442 dentists indicated that in 1943 fewer than 4 per cent employed dental hygienists. The proportion is highest in the New England area and lowest in the West-North Central area. Of the New England dentists, 8.6 per cent employed dental hygienists, while only 1.1 per cent of the West-North Central dentists employed them.

In "A Study of Incomes by Dentists in 1941," the American Dental Association Committee on Economics estimated that the number of hygienists per hundred dentists was 4.2, a figure closely in accord with Klein's.

According to an unpublished report of the American Dental Association Committee on Economics, in 1943 459 hospitals and related institutions employed 586 full-time hygienists; 435 hospitals employed 556 part-time hygienists; 204 military hospitals employed 550 full-time hygienists and nine employed twelve part-time hygienists. This would account for 1,700, or a little more than half of the estimated 3,000 in practice. The remainder are divided between schools and private practice.

Certainly, these figures indicate that the numerical growth of the dental hygienist has not kept pace with that of the dentist, the assistant or the technician; nor has it kept pace with the public's growing appreciation of dental service. A review of the past twenty-five years indicates that the dental hygienist movement reached a level about 1930. Since then, it has progressed very little, if any. How this auxiliary force can be better attuned to the practice of dentistry so that it can be utilized to permit dentists to broaden their service is a problem that soon must be solved by the profession. Its solution will require deep consideration of educational training to prepare hygienists for either prophylactic work or an educational career, or both. Furthermore, it will require consideration by the dentist as to how he can utilize the services of the hygienist in an endeavor to render greater service to the community. It seems that the trend in the dental hygienist movement during the past ten or fifteen years has been slightly downward rather than upward.

THE DENTAL TECHNICIAN

As the demand for dental service has grown and as the complexities of that service have increased, there has developed within dentistry a new craftsman or specialist known as the dental technician. Much has been said regarding the merits and demerits of the technician. The fact remains that the past fifty years has witnessed a tremendous growth in this auxiliary field.

The first commercial laboratory in this country was established about 1883 by Frank F. Eddy in Boston.[12, 13] Since that time, the number of laboratories and of technicians have greatly increased and, during recent years, the commercial dental laboratory has risen surprisingly in the respect of the profession. Most dentists in the latter part of the nineteenth century either did their

own laboratory work or were aided by an assistant who worked under their personal supervision. Today, it is estimated that more than 90 per cent of practicing dentists depend more or less on the commercial laboratories for their laboratory work.[12]

In 1890, there existed but two known commercial dental laboratories. At that time, there were 17,496 practicing dentists in the country, or one laboratory for 8,749 dentists. By 1920, there were approximately 100 laboratories for 56,152 dentists, a ratio of 1:562.[12] As of October 1 of this year, there were approximately 2,800 bona fide dental laboratories in the forty-eight states and the District of Columbia, a ratio of one laboratory to twenty-five dentists. The number ranges from a low of two each in Delaware and Nevada to 628 in New York. Pennsylvania has 255; California, 268 (data furnished by W. S. Rice, Coe Laboratory, based on figures supplied by the Dental Laboratory Institute of America, October 25, 1944); Illinois, 172. Twenty-five years ago, there were but thirty commercial dental laboratories in Illinois, only one or possibly two of which were located outside of Chicago.[15] (Information supplied by William Schroll, American Dental Company Laboratory, October 28, 1944.)

In 1941, there were about 15,000 technical workers employed by commercial dental laboratories. Since then, about 1,000 have been added to the ranks, but the war has withdrawn 7,500, or about 50 per cent, into the Army and Navy. These have been replaced by 4-F's and women. It is estimated that the Army and Navy will train approximately 10,000 technicians in the service. Thus, by the end of the war, there will be available from 32,000 to 35,000 dental technicians—almost one to every two dentists. It is extremely unlikely that all of these men, many of whom are but partially trained, will remain in this field after the war. In the postwar adjustment period, many will return to their former occupations. Others will seek new vocations. By a process of selection and elimination, only the better trained will be reemployed or retained by the commercial laboratories. In all probability, after the war, the number of technicians will return to slightly more than the 1941 figure; that is, to from 16,000 to 18,000, or one to every four dentists.

Another indication of the laboratory growth can be expressed in dollars. In 1920, the dental laboratories did a volume of business amounting to $15,000,000. By 1930, this had risen to $46,000,000 and, in 1943, it had increased to approximately $100,000,000. It has been estimated that the 1944 volume will reach $125,000,000. (Statement by William Schroll, American Dental Company Laboratory, October 28, 1944.)

When any auxiliary service reaches such size that its membership equals one-quarter the membership of the profession, and its gross income one-fifth of that of the profession, it is of sufficient importance to warrant the most serious consideration. Time and experience have demonstrated the practicability of dental laboratories and technicians. By employing the services of technicians, dentists have increased their ability to render service to larger numbers of people. However, the acceptance of the dental technician has placed added responsibilities on the dental profession. Chief of these responsibilities is that of guiding and regulating this auxiliary service so that the public will be protected from inferior appliances and unprofessional care.

In the past, little or no effort has been made to furnish dental technicians with a prescribed course of training. Laboratory operators have preferred to train their employes on an apprenticeship basis. Much credit must be given the leaders in the laboratory field for the advances made in technician training.

During the past twenty years, the quality of training has improved immeasurably. This is naturally reflected in the quality of appliances furnished the dentist for his patients.

Since the war, both the Army and the Navy have established technician training courses, and it may be possible that the establishment of these courses will be the forerunner of similar courses for civilian technicians in the postwar period. Certain it is that the dental technician, doing work that is an auxiliary branch of dentistry, is very likely to continue to serve in the future. The trend has been and continues to be upward.

COMMENT

In considering any dental health program for the people of the United States, whether it be on a national, state or local level, full consideration must be given to the problem of dental personnel. In the immediate postwar period, the United States will have somewhere between 70,000 and 75,000 dentists located largely in the urban areas. The services of these dentists will be augmented by the services of 35,000 assistants, 3,000 hygienists and perhaps 18,000 technicians. As Klein has pointed out, the ranks of the assistants could be increased 100 per cent, although, actually, a 50 per cent increase would probably reach the saturation point.

There is small likelihood of a noticeable increase in the number of hygienists in the immediate future. Neither is there likelihood of a perceptible increase in the number of technicians, as, under the present system of practice, one technician to every four dentists would seem to be almost sufficient.

We know from past experience that the public's appreciation of good dental health is increasing; we know that the demand for dental service is increasing, and we believe that the demand will continue to increase in the future. The problem that confronts the profession is how best to supply that demand with its present and foreseeable limited personnel.

A few men in the profession, and some outside the profession, have advocated extending the duties of both the dental hygienist and the technician so that they will be permitted to perform certain services now limited by statute to dentists. It is extremely doubtful that the dental profession will consider adopting these proposals unless it is willing to approve a double standard of dental service. In the light of the long years of struggle which dentistry has undergone in order to raise its professional standards to the present level, and in the light of results of similar experiments in England and Germany, it is very unlikely that the American people would accept a method of practice leading to a double standard of service.

CONCLUSION

How, then, with our limited personnel and with our limited knowledge of the etiology of dental caries, can the profession best meet the dental demands and

DENTAL HYGIENISTS

Recognized in dental practice acts of thirty-two states, although some are employed by institutions in forty-seven states and in every branch of the armed forces. Estimates of number in practice range from 3,029 to 7,000, 1,700 of whom belong to the American Dental Hygienists' Association through its twenty-six component societies. Fewer than 4 per cent of the dentists employ a hygienist. More than one-half of all hygienists are employed by schools, hospitals and other institutions.

The first school was established in 1913. Fifteen schools now offer courses. There was a total of 123 graduates in 1920, 293 in 1943.

ultimately the dental needs of the public?

It cannot be done overnight. It cannot be done by short cuts. It cannot be done by legislation.

As the National Health Program Committee and the Council on Dental Health of the American Dental Association have recommended, it can best be done by a program based on research, education and prevention or control. Once the etiology of dental caries is definitely determined and fully understood, effective preventive measures will undoubtedly follow. These measures will be more productive of enduring benefits than will efforts to delegate certain operative and restorative procedures to auxiliary personnel.

Until that day dawns when therapeutic measures can be employed to prevent dental caries, the dental profession should bend its every effort and the efforts of its auxiliary forces toward controlling caries by the most practical method known at the present time; namely, dental operative procedures for children. This is not a new formula. On the contrary, it is one that has been advocated for many years. Unfortunately, it has never been wholeheartedly accepted by the public, and it has been accepted only in principle, but not in fact, by many members of the profession.

In essence, this formula is contained in 3-b of the principles approved by the American Dental Association House of Delegates last month. It reads: "Programs developed for dental care should be based on the prevention and control of dental diseases. All available resources should first be used to provide adequate dental treatment for children and to eliminate pain and infection for adults."

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TOOTHACHE UNDER CONDITIONS SIMULATING HIGH ALTITUDE FLIGHT*

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INTRODUCTION

WAR in the air, whether as conducted by a fighter pilot or by a crew member, demands the utmost in personnel performance. The distraction of even a mild or intermittent toothache can seriously impair a flyer's efficiency. That toothache during high altitude flying presents a personnel problem in the Army Air Forces is demonstrated by its occurrence in 1.8 per cent of the cadets and officers given altitude training. The airman may have such severe pain that his only thought is of immediate and rapid descent.

To the dental officers in the Army Air Force, these high altitude toothaches present a problem of diagnosis and correction of the condition or eradication of the source of pain and, if possible, procedures aimed at prevention. The approach to a solution of these problems should include a correlation of clinical observations and histologic findings.

The medical aspects of selection and adequate care of flying personnel have so greatly expanded during the last few years that they now constitute a recognized specialty, grouped under the term "aviation medicine." The Army Air Force, through the operation of its school of aviation medicine at Randolph Field, Texas, is training its medical officers to the end that they shall be orientated in this newest medical specialty.

Dentistry in Aviation Medicine.—The following brief and elementary account of some of the medical aspects of aviation is intended as an introduction to any possible correlation of the basis of these toothaches and the mechanisms affecting other tissues in flight or decompression. Etiologically, general flying disorders may be grouped under five headings:

1. A severe vertigo brought on by the attempt at orientation in space. A conflict arises in visual, labyrinthine and somatic sensations.

2. Injuries from extremely low temperatures encountered in high altitudes.

3. An acute or chronic air sickness based on motion. This, in all its phases, resembles sea or train sickness, but is greatly intensified.

4. The diverse aspects of anoxia. Consideration of this group of disorders constitutes a large percentage of what is fast becoming a formidable literature on aviation medicine. The etiologic factor is a decrease in the partial pressure of oxygen incident to the decreased barometric pressure. Symptoms occur in the sphere of respiration, sight, muscular control, thought and memory processes. Prolonged anoxia may result in syncope or a severe, even irreversible shock. In the so-called decompression chamber, all flying personnel are given

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instruction in the need for and the utilization of oxygen.

5. Disorders due to the mechanical effects of decreased atmospheric pressure, including gastro-intestinal pain from distension, aero-otitis media, due to ventilation difficulties, severe injection of the bulbar conjunctiva, pressure or partial vacuum pains in the nasal accessory sinuses and a variety of disorders belonging to the group of aero-embolisms. The aero-embolic disorders are, presumably, due to the formation of nitrogen bubbles from body fluids and tissues at reduced barometric pressure. Almost any part of the body may be affected, according to the location of the bubble formation. The liberation of blood and tissue gases is thought to be on the same physical basis as are the compressed air disorders (caisson disease, or divers' paralysis, or bends), except that, in aviation, there is a decompression from one atmosphere to a fraction of one atmosphere, whereas, in caisson disease, there is a decompression from pressures above normal to one atmosphere.

The five possible etiologic factors will be discussed with regard to toothache in high altitude flight.

THE LITERATURE

Armstrong's "Aviation Medicine"[1] presents the most comprehensive discussion of this field, without, however, mentioning dental problems. The "Bibliography of Aviation Medicine," by Hoff and Fulton,[2] complete to May 31, 1942, lists in its 200 pages less than a dozen references to reports dealing with dental tissues in high altitude flying. Most of these early dental publications deal with oral hygiene for flyers and the use of dental models in identification. One report was entitled "The Loss of Dental Fillings by Aviators in High Altitude Flights." There were some reports on the loss of the intermaxillary dimension in relationship to earache developed in flying, and one pointing out the dangers in the use of removable restorative appliances for pilots.

With the adoption of the use of decompression chambers in the training of flying personnel in Army and Navy Air Forces, the number of persons subjected to simulated high altitudes was greatly increased. Among the 1,800 or more articles written on aviation medicine since June 1942, there are numerous dental reports. In the more recent publications, it is evident that most writers have recognized the problem confronting dentistry and are attempting to explain it on a physiologic basis.

The following review is confined to the dental literature dealing with toothache occurring at reduced barometric pressure, since the investigation did not extend to the consequences of an inadequate vertical dimension. A considerable amount of recent work cannot be quoted until released from wartime rulings. Thus, the following is a summary of pertinent literature now available.

In 1933, Neblett[3] wrote on aerodontia, stating that apical abscesses and chronic periodontoclasia, separate or combined, were not unusual causes of "staleness" in pilots. Neblett cited nine cases in which immediate improvement in neurocirculatory disturbances followed elimination of oral infection. No mention was made of actual toothache in flight.

In 1937, Dreyfus[4] reported a painful pulpitis at an altitude of 6,100 feet, which he relieved by pulpectomy. In the same year, Armstrong and Huber[5] concluded, after experimenting on extracted teeth, that neither oxygen lack and cold nor atmospheric pressure, nor any combination of these conditions caused any evident injury to teeth or dental restorations.

Levy,[6] in 1942, quoting from foreign letters to the *Journal of the American Medical Association,* stated that poorly filled teeth ache at altitudes above 25,000 feet. He also speculated on the possibility of pain from expansion of air spaces in

teeth with improperly filled root canals. He refers to a statement by Koelsch that inhaling cold air or oxygen may loosen fillings owing to differences in the expansion of tooth substance and filling material.

In the *British Dental Journal* of September 1942, there is a report stating that many cases of pain of dental origin had occurred in both the British and the Swiss air forces, some being relieved by extraction, others by devitalization or apicoectomy. This report was quoted by Willhelmy.[7]

In September 1942, Lipson and Weiss[8] emphasized the need of a biologic approach to the dental aspects of aviation medicine. Their speculations included a "blackout" within the tooth from the centrifugal force in flying maneuvers, and the possible effects of prolonged vasoconstriction due to extreme cold. The possibility of pain in the pulp, due to aero-embolism, was also taken into consideration.

In 1943, Joseph *et al.*[9] reported on a six-months experience with dental pain during decompression tests at the Naval Air Station at San Diego, Calif. The investigators recorded a 1.2 per cent incidence, and concluded that pain was due either to the presence of carious teeth with vital pulps or teeth with degenerated gangrenous pulps, attributing this to the change in atmospheric pressure. They also listed the expansion of air trapped under an inlay as a possible source of pain. The writers, who advocated great care in preoperative diagnosis, stated that a maxillary sinusitis should be included in the differential diagnosis. Among their cases were two in which painful symptoms arising from carious teeth were relieved by the use of protective cement bases, and two in which pain was attributed to teeth with gangrenous pulps. It was concluded that pain of dental origin in the low pressure chamber may, at times, assist in disclosing hidden degenerative or carious processes. This statement on caries was taken up by the daily press and given wide publicity.

Willhelmy,[7] in July 1943, reviewed the literature and reported a case that he had in 1935 in which two teeth causing pain at an altitude above 8,000 feet were freed from symptoms by opening the pulp chambers. The teeth were subsequently extracted.

Coons,[10] in July 1943, reported three cases of chronic periapical infection which became acute and painful as a result of decompression. He mentioned the case of a British flying officer who, because of dental pain, was forced to descend to the ground when on a flying mission. Coons advocated elimination of all potential periapical and gingival foci in flying personnel. He considered the possibility that aero-embolism or nitrogen expansion might be a cause of high altitude toothache, and he recommended universal employment of protective bases under all fillings.

In 1944, Mitchell,[11] in a paper entitled "Aerodontalgia," reported his experience with twenty-five cases of high altitude toothache at Randolph Field, Texas. He attributed pain to the presence of carious teeth, teeth with deep restorations and teeth having periapical involvements. Treatment consisted of extraction of abscessed teeth and, for vital teeth, the use of zinc oxide and eugenol, either as a temporary filling or as a base under amalgam fillings. He reported good results with each of the five cases under observation. In his summary, Mitchell stated that the cause is undetermined, although it appears to be the result of either local anoxia or aero-embolic conditions, or a combination of the two. He referred to Armstrong for the latter explanation and to Knisely, of the University of Chicago, for the local anoxia theory. Mitchell recommended a more frequent use of protective bases under large fillings.

In a naval publication, in January

1944, Restarski[12] reported work on extracted teeth in which he was unable to dislodge improperly manipulated fillings, or fillings with air spaces, by subjecting the teeth to low barometric pressures.

In 1944, Kennon and Osborn[13] published a report on a series of 175 cases, concluding that pain, during decompression, is one of the earliest subjective symptoms of pulpitis, and that, with appropriate treatment, many early cases of pulpitis can be corrected. They found that the use of zinc oxide and eugenol is highly successful in the treatment of mild pulpitis, and that all deep fillings and pulp cappings for flying personnel should be checked by altitude tests. They suggest special instruction in diagnosis, prevention and treatment of aerodontitis for all dental officers treating flying personnel.

Harvey[14] found that low temperature in high altitude flying was a negligible factor in pain.

MATERIAL AND METHOD OF INVESTIGATION

The teeth employed in this study were extracted from a group of patients, all healthy males between the ages of 18 and 27, referred to the dental clinic from an altitude training unit between January 1943 and April 1944. During this period, about 250 cases were observed clinically and, from this group, seventy-five teeth were prepared for histopathologic study.

Clinical examinations consisted of case history, visual inspection, percussion, exploration, roentgenographic examination and tests for response to ice. The cases thus observed included intact teeth and teeth presenting caries of varying degree, obvious pulp involvement and nonvital pulps. Some experimental fillings were placed, and repeated altitude tests made in many cases. Most of the men did not actually experience high altitude flights, but were subjected to simulated conditions in a decompression chamber, a device in which flying personnel can be put under barometric pressure reductions corresponding to those experienced in flight up to an altitude of 38,000 feet. The barometric pressure is reduced to about one-half of that at sea level at 18,000 feet; to about one-third at 27,000 feet, and to about one-fifth at 38,000 feet.[15] The 38,000 foot level represents about the ceiling of present-day combat flight.

The seventy-five teeth selected for histologic investigation were fixed either in 10 per cent neutral formalin or in freshly mixed Zenker formalin solution (Maximow). Immediately after extraction, the teeth were ground on two sides so that only a thin layer of dentin covered the pulp. Thus, the fixing solution could penetrate the pulp tissue at once. After decalcification, the teeth were embedded in celloidin and sectioned. The sections were stained with hematoxylin-eosin or other dyes, according to special needs. For comparison of the findings obtained in these seventy-five teeth, a group of 210 teeth extracted at different dental schools or in private practice during the past six years were used. These 210 teeth were fixed and prepared in the same manner.

To evaluate the findings, it was necessary to compare pulps of teeth extracted owing to pain at lowered atmospheric conditions with those extracted under ordinary conditions. An attempt had to be made to determine what changes took place in the pulps before they were subjected to lowered atmospheric pressure, and to determine what changes might be attributed to the effects of lowering the atmospheric pressure.

CLINICAL AND HISTOPATHOLOGIC FINDINGS

The first case of pain at simulated high altitude occurred shortly after installation of the altitude training unit. The patient had pain in the region of the lower left lateral incisor at 25,000 feet. Clinically and roentgenographically, the teeth appeared normal except for an

indication of stones in the pulp chambers. Although a repeated test in the decompression chamber showed identical results, the patient was dismissed without diagnosis or dental treatment. Attempts at diagnosis failed in many of the early cases because the search was for some obvious pathologic process, such as periapical lesions, nonvitality, caries or the presence of defective fillings.

Later, attention was focused on recently filled teeth with vital pulps, which,

Fig. 1.—P222: Painless ascent to 38,000 feet: Normal pulp (*P*); large pulp stone (*PS*) in pulp chamber; small pulp stones (PS_1) close to nerve bundles (*N*); *D*, dentin. (Upper second molar.)

on application of ice, reacted with continuous pain. When such teeth were extracted, the patient had no pain at 38,000 feet.

REPORT OF CASES

The condition of the seventy-five teeth sectioned and stained for this investigation was diagnosed clinically and the teeth were extracted because of pain in the decompression chamber. In addition, some teeth were studied because of treatment problems involved.

The cases to be presented are arranged according to the predominant histologic finding in the pulp; namely, normal pulp tissue, edema, acute inflammation, chronic inflammation and nonvitality.

Normal Pulps.—In our material, the pulps that were closest to normal were P222, P228 and P249. In P222, an upper second molar, a normal pulp had been instrumentally exposed. (Fig. 1.) In the decompression chamber, the patient encountered no pain up to 38,000 feet. P249, a right upper second bicuspid, had a small amalgam filling on the mesial side with some soft dentin below the filling. The amalgam was removed and, in excavating the cavity, the pulp was instrumentally exposed. Clinically, the pulp was considered normal. The cavity was left open and the patient was sent to the decompression chamber immediately. There was no discomfort at 38,000 feet. The tooth was extracted half an hour after descent. Histologic investigation revealed a normal pulp structure with a slight edema in one pulp horn. P228, a lower bicuspid, differed clinically from P249 in that it had a large carious cavity. The tooth was very sensitive to cold and heat, but there had been no unprovoked pain. At the time of cavity preparation, the floor of the cavity appeared pink and was very sensitive to touch and thermal irritation. Clinically, the tooth was hypersensitive. Without any attempt at therapy, the patient was sent into the decompression chamber for a test, and no pain was encountered up to 38,000 feet. The tooth was extracted one half hour after return to ground level.

The histologic picture revealed the floor of the cavity to be within the irregular dentin in the pulp horn. (Fig.

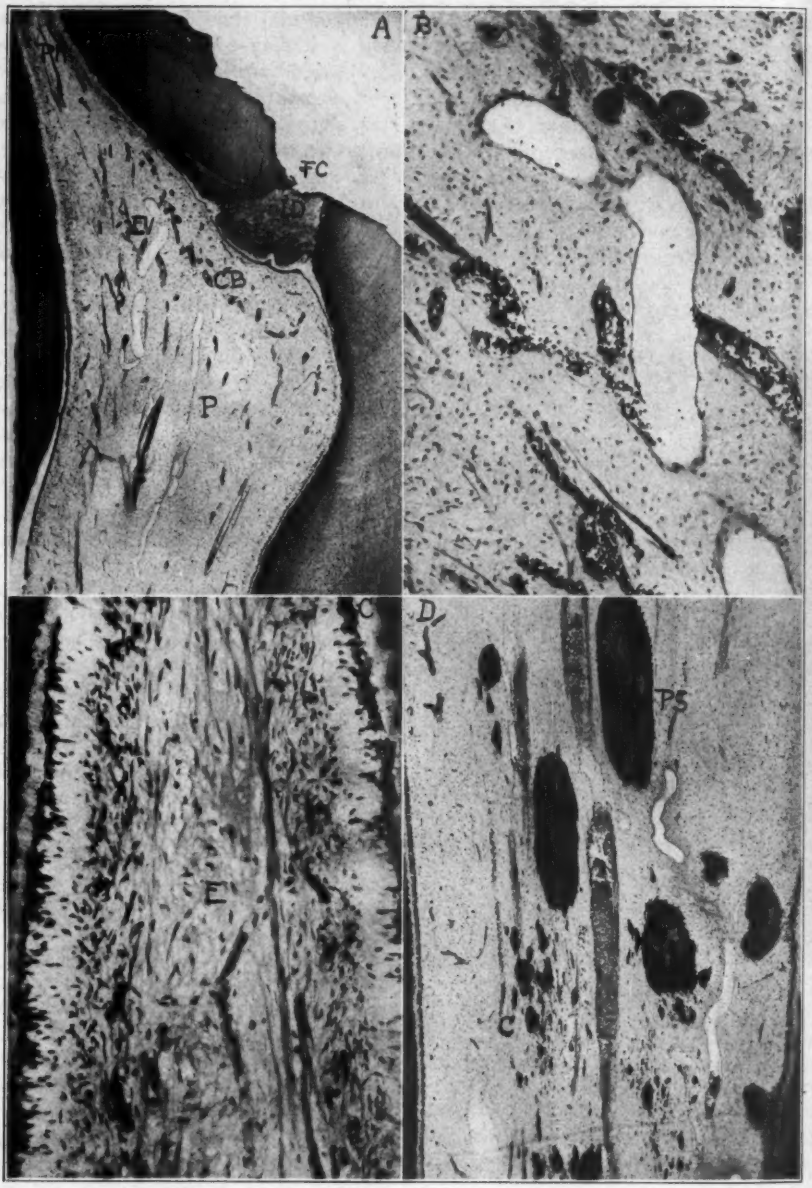

Fig. 2.—P249: Painless ascent to 38,000 feet: *A*: General view showing floor of cavity (*FC*); *ID*, irregular dentin below floor of cavity; *P*, pulp; *PH*, pulp horn; *EV*, empty blood vessels; *CB*, congested blood vessels. *B*: Higher magnification of area *EV*; blood from a larger vessel pressed into side branches. *C*: Higher magnification of pulp horn; slight edema (*E*). *D*: Root canal with pulp stones (*PS*) and calcification (*C*). (Lower bicuspid.)

2*A*.) The irregular dentin filled the entire pulp horn, and thus exposure of the pulp in the preparation of the cavity was prevented. Below the irregular dentin, the odontoblasts were shortened, but regularly arranged. The area of uncalcified predentin was somewhat wider than that of the regular dentin, and calcification was irregular and not quite complete. The blood vessels were greatly congested in the upper part of the pulp. Some of the veins in the area just below the con-

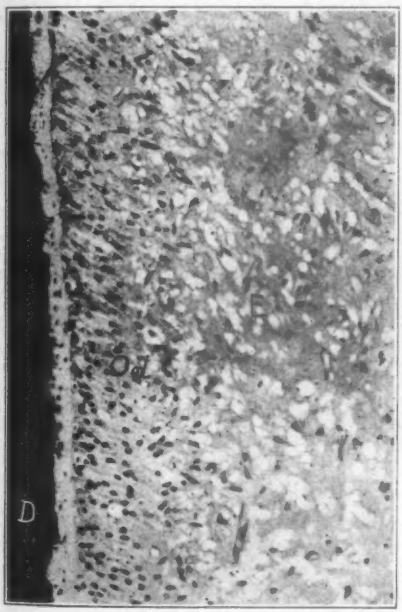

Fig. 3.—P284: Sharp pain at 24,000 feet: Edema (*E*) in entire pulp reaching between odontoblasts (Od), disrupting regular arrangement; *D*, dentin. (Upper second molar.)

gested vessels were empty and dilated. It seems that the blood had been forced from the larger into the smaller veins. (Fig. 2*B*.) In some vessels, the blood cells were tightly packed, the condition simulating hyalin thrombus formation. The tissue in the pulp horn was edematous (Fig. 2*C*), the edema reaching between the odontoblasts. There were only a few inflammatory cells of the lymphocytic type. In the root canal of the tooth were numerous smaller and larger pulp stones. (Fig. 2*D*.) The connective tissue of the pulp in this area showed a slight degree of hyalin degeneration. Calcium was being deposited in the hyalinized tissue. Some of the blood vessels were filled with blood, while others were empty.

The histologic investigation of this case revealed that, prior to cavity preparation, caries had caused the irregular dentin formation. Preparation of the cavity in this irregular dentin was responsible for the pink appearance of the dentin at the floor of the cavity. No inflammatory reaction of any serious degree was observed.

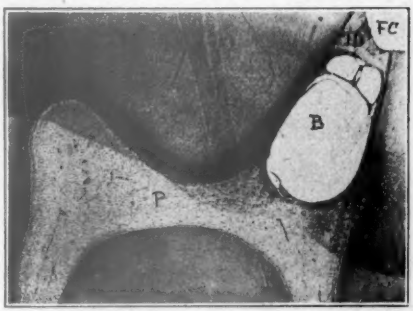

Fig. 4.—P258: Pain at 2,000 feet; intolerable at 10,000 feet: Large bubbles (*B*) in pulp horn; *P*, pulp; *ID*, irregular dentin; *FC*, floor of cavity cut into irregular dentin. (Lower molar.)

The three cases differ only in their vascular reaction. The pulp tissue itself appeared to be normal. The proximity of the cavity to the pulp in P228 might be responsible for some of the vascular damage and hyperemia in the peripheral vessels. The congestion in this case was further aggravated by the mechanical effects of decompression. The open cavities and absence of inflammatory reaction are assumed to account for freedom from pain during the decompression tests.

Teeth with Edematous Pulps.—Of the

seventy-five sectioned teeth, sixteen presented a pathologic picture of a typical edema of various degrees. To illustrate this group, three cases will be described. P284, an upper second molar that was filled with amalgam several months previously, never caused any pain. In the decompression chamber, the patient felt sharp pain at 24,000 feet, which was relieved on descending to 21,000 feet. In the clinic, the tooth was not sensitive to percussion, but responded with pro-

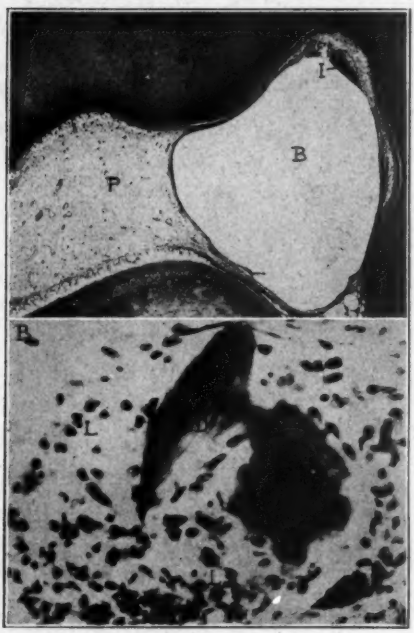

Fig. 5.—P223: Pain at 5,000 feet; intolerable at 10,000 feet: *A*: Large bubble (*B*) in pulp horn; acute inflammation (*I*) in tip of pulp horn; *P*, pulp. *B*: Higher magnification of region *I*; dentin splinters (*D*) surrounded by polymorphonuclear leukocytes (*L*). (Lower second molar.)

longed pain on irritation with ice. Histologically, the pulp showed extensive edema (Fig. 3) without any inflammatory cells. There was no hyperemia or any other pathologic change in the pulp.

P258, the second case representing this group, was a lower molar that became painful in the decompression chamber at 2,000 feet. The pain persisted with increasing intensity, becoming intolerable at 10,000 feet. Clinically, there was no pain on percussion, but the tooth was sensitive to ice, and the pain persisted for some time. Histologically, the pulp showed considerable edema without any inflammatory cells. The most striking feature of this specimen was a large empty space in one pulp horn. (Fig. 4.) The pulp tissue was compressed at the periphery of the pulp chamber. The presence of a few red blood corpuscles in this area indicated that the bubble formation was of recent and vital origin.

The third case of pulp edema, P223, was a lower second molar filled with amalgam three weeks previously. The tooth was sensitive to cold and was the source of a few sharp, unprovoked pains. In the decompression chamber, pain started at 5,000 feet and became intolerable at 10,000 feet. It was relieved on descent to 4,000 feet. The histologic section (Fig. 5*A*) showed an edematous pulp with a large empty space in one horn, extending through the pulp chamber to the beginning of the root canal. Toward the pulp horn, a few dentin splinters could be seen surrounded by some polymorphonuclear leukocytes and lymphocytes. The presence of the dentin splinters indicated that, at the time of cavity preparation, the pulp was exposed and the dentin splinters were pushed into the pulp. The inflammatory reaction was mild and localized around the dentin splinters. (Fig. 5*B*.) The rest of the pulp showed considerable edema, but was free from all inflammatory cells. An area of acute hemorrhage could be seen surrounding the bubble.

In most of the sixteen cases with edematous pulps, there were no inflammatory cells in the tissue. Rarely, a few disseminated inflammatory cells were found. The pain level of the sixteen

cases ranged from 2,000 to 25,000 feet, with an average of 11,000 feet.

Teeth with Acute Pulpitis.—The histologic material included seventeen teeth with acutely inflamed pulps. The nine selected for presentation were arranged in a sequence from the slightest to the most severe degree of acute inflammation.

P221 was an upper first molar filled with amalgam one week before extraction. It was sensitive to both hot and cold, and was painful in flying above 2,000 feet. On the day of extraction, there was pain in the decompression chamber at 2,000 feet. The patient was forced down by pain at 24,000 feet. Pain stopped at ground level. In the clinic, thirty minutes later, the tooth proved sensitive to cold and slightly painful to pressure. The roentgenogram revealed an area of radiolucency under the filling. Removal of the filling disclosed carious dentin covering the pulp horn.

Histologic investigation showed the cavity to be very close to the pulp. (Fig. 6*A*.) Irregular dentin filled the pulp horn. Almost the entire pulp chamber was occupied by a large pulp stone, attached to the floor and also to the roof of the pulp chamber. There was extensive calcification also in the root canals where the connective tissue showed hyalin degeneration. Some of the calcified bodies were large pulp stones, others fine amorphous precipitates.

In the pulp horn, close to the floor of the cavity, the odontoblasts were irregularly arranged, being separated from

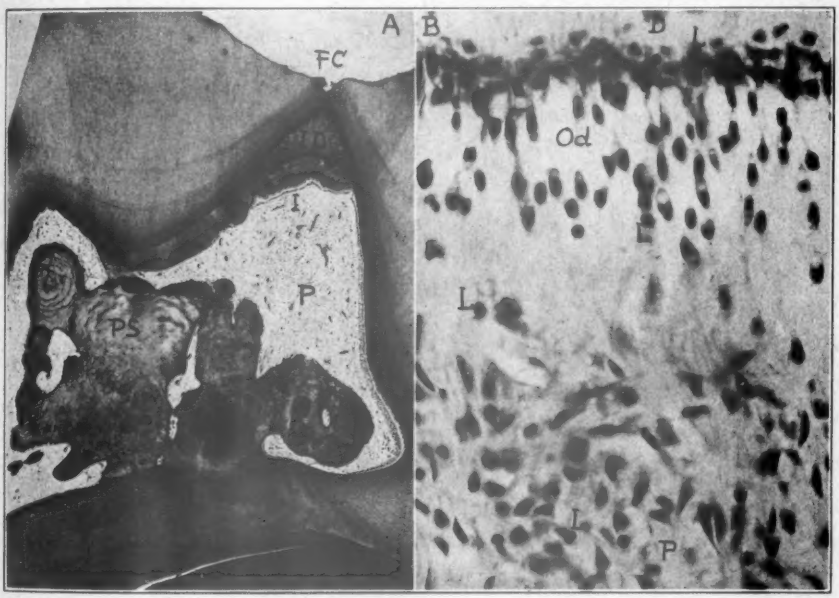

Fig. 6.—P221: Pain at 2,000 feet: *A*: General view showing large irregular pulp stone (*PS*) in pulp (*P*); irregular dentin (*ID*) in pulp horn; floor of cavity (*FC*); slight acute inflammatory reaction (*I*) in pulp horn. *B*: Higher magnification of area *I* showing odontoblasts (*OD*) with intercellular and intracellular edema. Polymorphonuclear leukocytes (*L*) are scattered through the pulp (*P*) and odontoblasts. (Upper first molar.)

each other by intercellular edema, and showed also a considerable degree of intracellular edema. (Fig. 6*B*.) There were a few polymorphonuclear leukocytes between the odontoblasts, as well as deeper in the pulp tissue. These cells were not accumulated in large masses or in groups, but were scattered in the entire pulp horn. A few mononuclear lymphocytic cells were also present. This tissue reaction is indicative of a moderate subacute inflammatory reaction. A certain amount of congestion was indicated by the presence of highly filled blood vessels close to the pulp horn. The large stone in the pulp chamber and the degeneration and calcification in the root canals must have caused considerable circulatory difficulty. In correlating the clinical history with the histopathologic findings, we may assume that a carious process had been active in this tooth for a considerable time before the filling was placed. This was indicated by the amount of irregular dentin formed in the pulp horn and at the roof of the pulp chamber. There was no indication that the large pulp stone had any relation to the carious process. It is probable that its presence was merely incidental.

The high degree of irritability of this tooth, i.e. pain at 2,000 feet and from heat and cold and on percussion, was probably primarily due to the acuteness of the inflammation and to circulatory difficulties in the pulp because of the

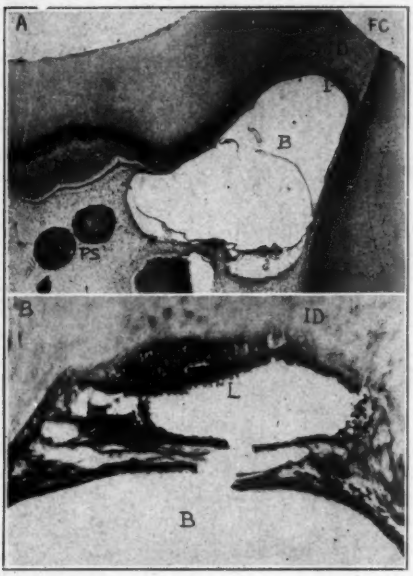

Fig. 7.—P213: Pain at 6,000 feet; intolerable at 12,000 feet: *A*: General view showing large bubble (*B*) in pulp; I, acute inflammatory reaction area; *ID*, irregular dentin; *FC*, floor of cavity; *PS*, pulp stones. *B*: Higher magnification of area *I*; accumulation of polymorphonuclear leukocytes (*L*) forming small abscess in pulp horn; *B*, bubble; *ID*, irregular dentin. (Lower first molar.)

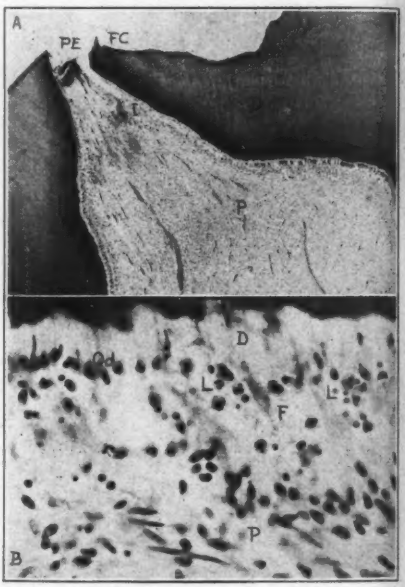

Fig. 8.—P242: Pain at 10,000 feet: *A*: General view showing pulp exposure (*PE*) at tip of pulp horn; *FC*, floor of cavity; *I*, inflammation; *P*, pulp; *D*, dentin. *B*: Higher magnification of area *I*; polymorphonuclear leukocytes (*L*) between odontoblasts (*Od*); edema with precipitation of fibrin (*F*); *P*, pulp; *D*, dentin. (Upper third molar.)

stones in the pulp chamber as well as in the root canals. This tooth, we may assume, would have become painful sooner or later regardless of exposure to lowered atmospheric pressure. The acute inflammatory reaction indicated that infectious material, toxins or bacteria, from the carious cavity had reached the pulp. The exposure of the pulp to low atmospheric pressure placed a burden upon the circulation which it could not carry.

P213 was a lower first molar filled with amalgam eight weeks earlier because it had become sensitive to sweets. There was no pain after the filling was placed, but the pilot could not fly above 6,000 feet without having pain in this tooth. On the day of extraction, an intolerable toothache forced him to dive his plane from 12,000 feet. In the clinic, the tooth was not sensitive to percussion, but was persistently sensitive to irritation with ice. The tooth was extracted immediately.

Histologic investigation revealed a slight acute inflammatory reaction in the pulp horn. The most striking finding in this specimen was the large empty space extending over the entire pulp horn. (Fig. 7*A*.) The tissue was compressed toward the pulp chamber and considerable hemorrhage could be seen in this area. In the tip of the pulp horn, an ac-

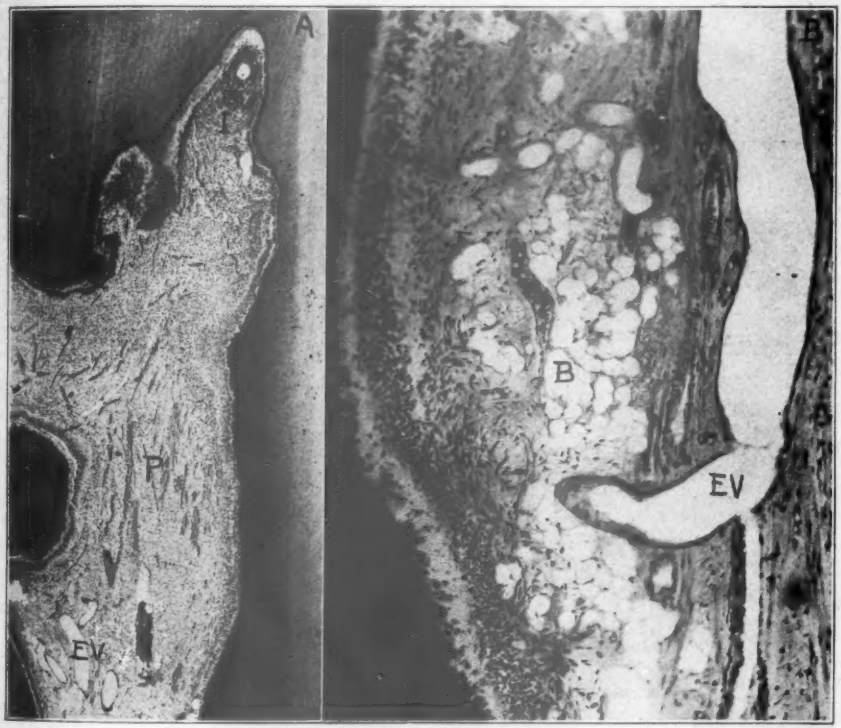

Fig. 9.—P260: Pain at 5,000 feet; intolerable at 6,000 feet: *A*: Acute inflammatory reaction (*I*) in pulp horn. The pulp (*P*) is somewhat hyperemic. Some blood vessels are dilated and empty (*EV*). *B*: Higher magnification of area *EV*. There is bubble formation (*B*) in close proximity to a dilated empty blood vessel (*EV*). (Upper first bicuspid.)

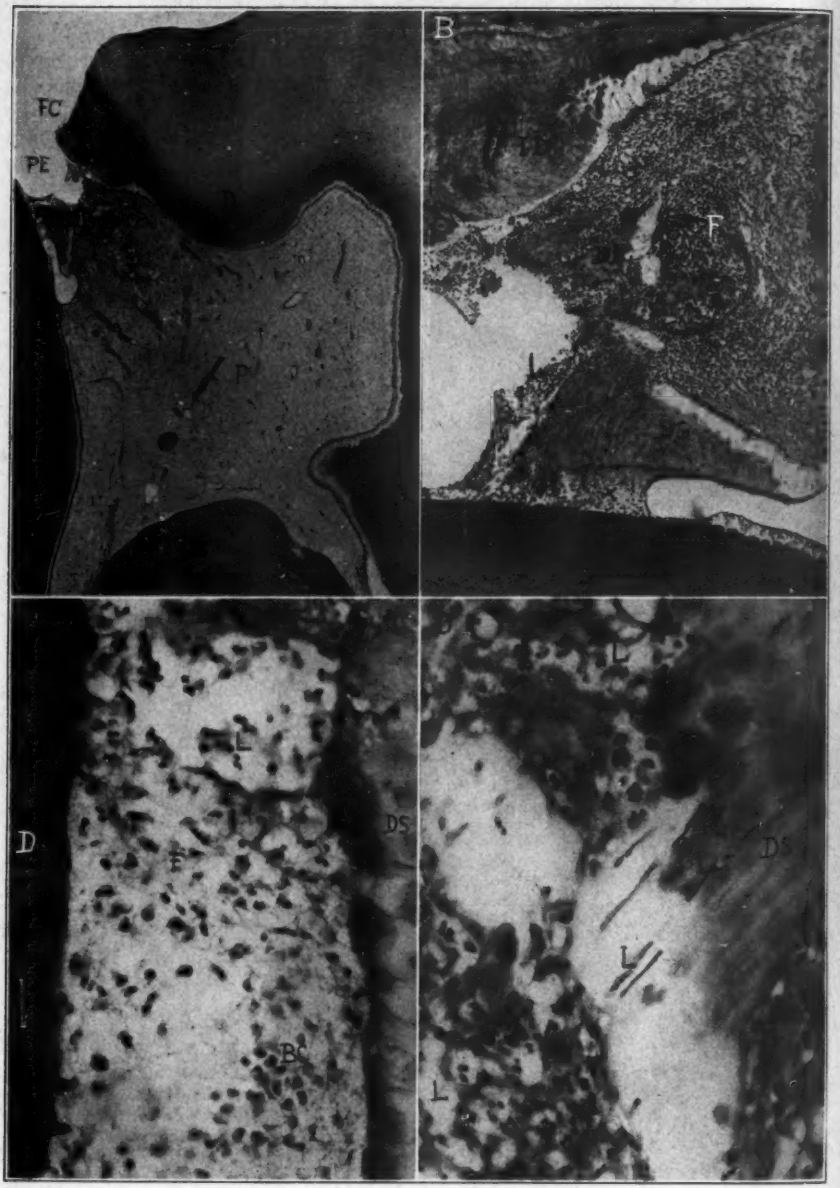

Fig. 10.—P233: Pain at 11,000 feet: *A*: General view showing pulp exposure (*PE*) at one pulp horn; floor of cavity (*FC*); irregular dentin (*ID*) close to exposure area; acute inflammatory reaction (*I*) in pulp horn; *P*, pulp; *D*, dentin. *B*: Higher magnification of area *I*; dentin splinters, (*DS*) scattered in pulp, surrounded by polymorphonuclear leukocytes (*L*) and fibrin (*F*); *P*, pulp; *ID*, irregular dentin. *C*: Higher magnification of area *X* (view *B*), showing fibrinous network (*F*) with polymorphonuclear leukocytes (*L*) and red blood cells (*BC*); *D*, dentin; *DS*, dentin splinter. *D*: Higher magnification of *DS* (in *B*); dentin splinters (*DS*) invaded and surrounded by polymorphonuclear leukocytes (*L*). (Upper first molar.)

cumulation of leukocytes formed a small abscess. (Fig. 7*B*.) Several small pulp stones were in the pulp chamber and the rest of the pulp chamber showed signs of edema. There was considerable irregular dentin formation in the pulp horn as well as on the roof of the pulp chamber corresponding to the cavity.

P242 was an upper left third molar. The patient had been in the low pressure chamber up to 28,000 feet and had done considerable high altitude flying without having any toothache. The tooth was filled with amalgam the day before extraction. On the same day, pain was felt at 2,000 feet, subsiding somewhat, but becoming acute at 4,000 feet and again at 6,000 feet. The next day, pain recurred at 10,000 feet. It subsided somewhat, but the flyer was forced to descend from 18,000 feet. He reported at the clinic immediately after landing. The tooth was sensitive to percussion and hypersensitive to cold. The filling was removed and the pulp found to be exposed.

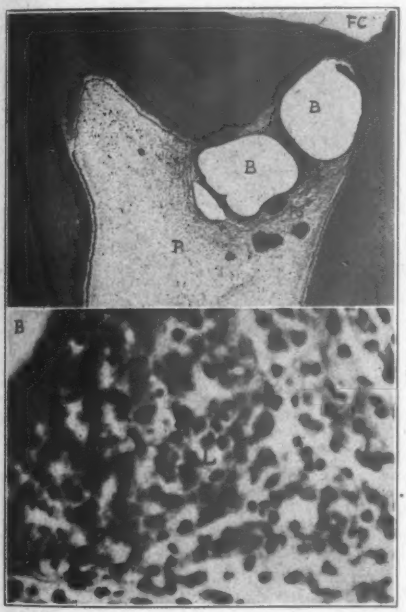

Fig. 11.—P265: Intolerable pain at 5,000 feet: *A*: General view showing large bubbles (*B*) in pulp horn surrounded by area of acute inflammatory reaction (*I*); *PS*, pulp stones; *P*, pulp; *FC*, floor of cavity. *B*: Higher magnification of area *I* (view *A*), showing polymorphonuclear leukocytes (*L*). (Upper first molar.)

Histopathologic investigation revealed an exposed pulp horn without any irregular dentin formation in this area. (Fig. 8*A*.) The pulp tissue in this region showed considerable inflammatory exudate and some hemorrhage. The odontoblastic layer below the floor of the cavity was disarranged and considerable edema was found, with precipitation of fibrin in the exudate. (Fig. 8*B*.) In the remaining pulp, there was some edema without extensive hyperemia.

Correlation of the clinical and histopathologic investigations readily explains the findings. The tooth had a cavity, but the carious process had not as yet affected the pulp tissue. No pulp irritation was present before cavity preparation, a fact which explains the absence of irregular dentin in the pulp chamber. Presumably, there had been a layer of sound dentin between the carious cavity and the pulp, and the exposure occurred accidentally during cavity preparation. The irritation set up by this procedure had the acute inflammatory reaction seen in the microscopic specimen. The pain symptom is explained by acute pulpitis and hyperemia, further aggravated by the low barometric pressure.

P260 was an upper first bicuspid in which an amalgam filling had been placed three weeks earlier. There had been no pain before the filling. The following night, the patient had a few sharp pains, and, for the next week, the tooth was hypersensitive to thermal irritation. In the decompression chamber, the pain was sharp at 18,000 feet. In the clinic, the tooth was very sensitive to ice and continued to cause pain, but was not

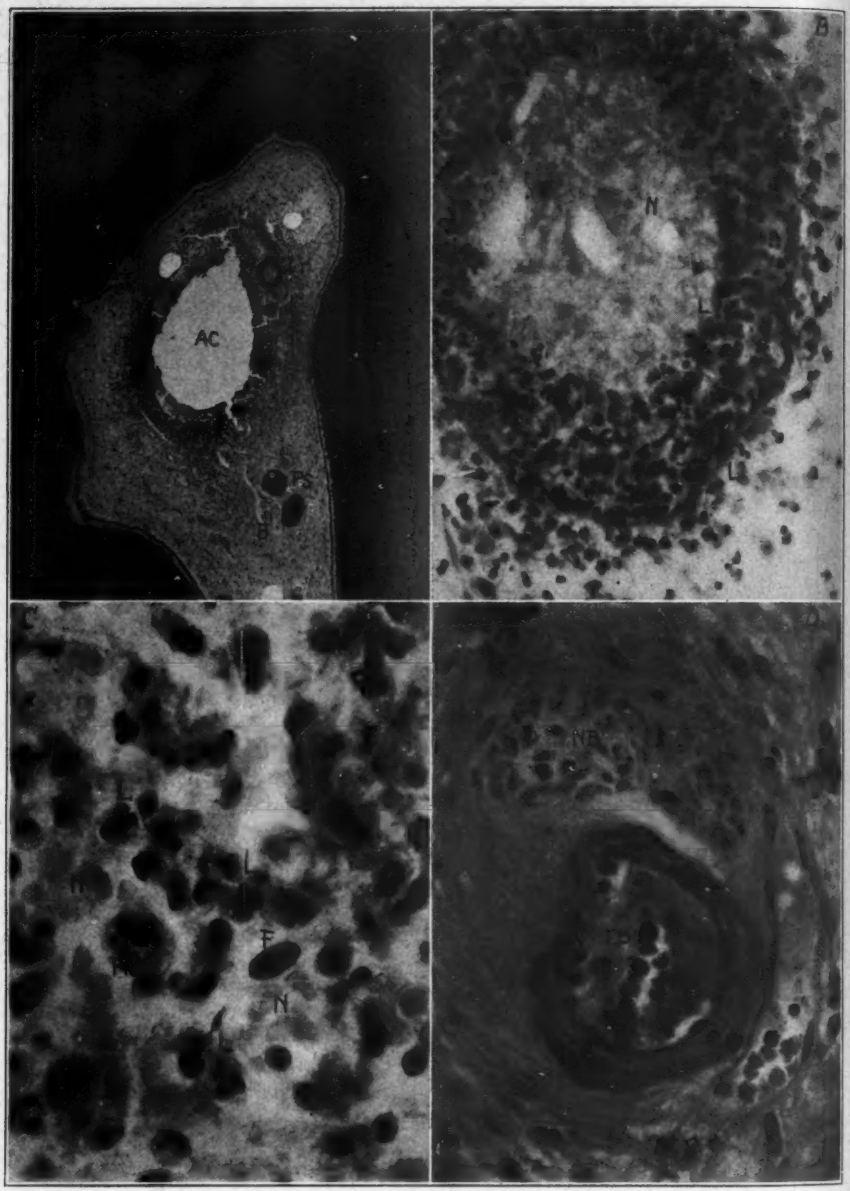

Fig. 12.—P245: Pain at 6,000 feet: *A*: Abscess cavity (*AC*) in pulp chamber surrounded by polymorphonuclear leukocytes. In some areas, smaller abscesses are in formation (*I*). *PS*, pulp stones; *B*, thrombosing blood vessel. *B*: Higher magnification of *I* (view *A*); accumulation of polymorphonuclear leukocytes (*L*) surrounding necrotic center (*N*). *C*: Higher magnification of polymorphonuclear leukocytes (*L*) and phagocytes (*Ph*) mingled with necrotic tissue material (*N*). *D*: Higher magnification of area *B* (in *A*); thrombosed blood vessel (*TB*); *NB*, nerve bundle. (Upper second molar.)

sensitive to percussion. The amalgam filling was removed and the pulp was found exposed. The cavity was painted with cavity varnish and a filling of zinc oxide and eugenol was placed. The patient returned to the decompression chamber and, at 5,000 feet, felt pain, which became intolerable at 6,000 feet. The tooth was extracted in the decompression chamber at 6,000 feet (the only tooth extracted in the decompression chamber). Pain did not return on repeated decompression tests.

Fig. 13.—P286: No flight: Acute abscess (*A*) in pulp chamber; *DS*, dentin splinters; *ID*, irregular dentin. (Upper cuspid.)

Histologic investigation revealed an acute inflammation in the pulp horn (Fig. 9*A*) with some hyperemia and congestion of the peripheral blood vessels. Near the floor of the pulp chamber, some wide, dilated and empty veins could be seen. (Fig. 9*B*.) In close proximity to these wide blood vessels, numerous empty spaces were formed, resembling fatty tissue. That this was not fat tissue is evident from the lack of the typical signet-ring effect. No inflammatory cells could be seen in this area.

P233 was a right upper first molar with a deep cavity. The patient came to the clinic free from pain. Careful excavation disclosed that the carious process had reached the pulp and a bleeding pulp horn was exposed. For experimental purposes, tinfoil was placed over the area of exposure and an amalgam filling was made. One hour later, the patient was sent into the decompression chamber. He had a toothache at 11,000 feet and again at 18,000 feet, and the pain persisted from 28,000 to 33,000 feet. At ground level, the pain was reduced by about half. The tooth was extracted one hour after descent.

The histopathologic finding in this case was that of an acute inflammatory reaction in the pulp horn where the pulp was exposed. (Fig. 10*A*.) There was a considerable amount of irregular dentin formation in the pulp horn, an indication of previous irritation in this area. There were a few splinters of dentin within the infiltrated pulp tissue. (Fig. 10*B*.) The infiltration was most extensive in the pulp horn and was limited toward the rest of the pulp tissue by a belt of dilated, hyperemic blood vessels. The tissue in this area was edematous. The cellular elements of the inflammatory infiltration consisted exclusively of polymorphonuclear leukocytes. In some areas, a fibrinous network had been infiltrated by the leukocytes. (Fig. 10*C*.) Red blood corpuscles were also seen within the fibrin network. In other areas, the leukocytes were densely packed and could be considered as a beginning abscess formation. The area was surrounded by a fibrin capsule. The leukocytes were

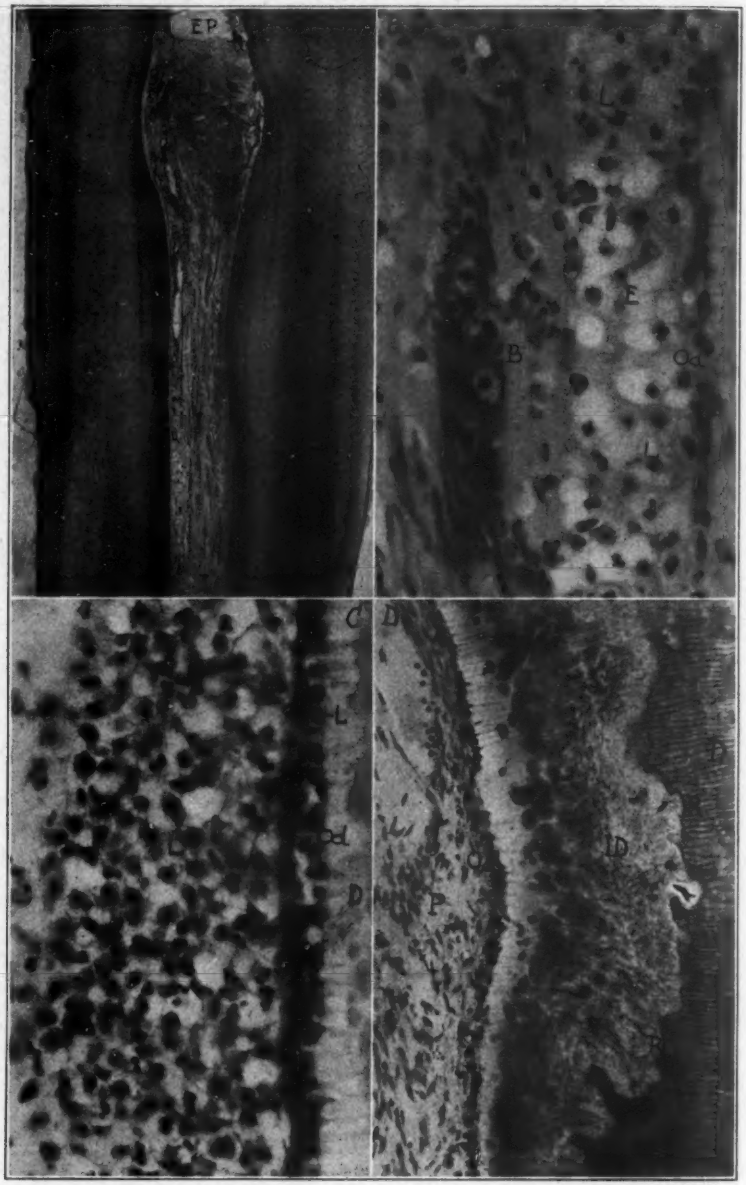

Fig. 14.—P226: Pain at 11,000 feet: *A*: General view showing total pulpitis; *EP*, pulp exposure; *I*, acute inflammation. *B*: Higher magnification of inflammation area in root canal; stasis in blood vessel (*B*); polymorphonuclear leukocytes (*L*) in fibrinous, edematous (*E*) area; odontoblasts (*Od*). *C*: Higher magnification of inflammation area in root canal; large number of polymorphonuclear leukocytes (*L*) in fibrinous network and between odontoblasts (*Od*) and dentin (*D*). *D*: Repaired area of resorption close to apex in root canal; *P*, pulp; *Od*, odontoblasts; *ID*, irregular dentin; *R*, line of resorption; *D*, dentin. (Upper lateral incisor.)

found not only between the dentin splinters in the pulp tissue, but also in the dentinal tubules of the dentin splinters. (Fig. 10*D*.)

The correlation of the clinical with the histopathologic observations presents some problems. The inflammatory reaction was of an entirely acute nature, no chronic inflammatory cells being present. Nevertheless, a considerable amount of irregular dentin formation pointed to a chronic irritation in this area at one time. This irritation may, in its latest stages, have brought about the acute inflammatory reaction. The question arises as to whether the mechanical exposure of the pulp, whereby some of the infected dentin splinters were pressed into it, was mainly responsible for the inflammatory picture. The presence of irregular dentin on the one hand, and the acuteness of the inflammatory reaction on the other hand, would indicate that an inflammation might have been present before the pulp was exposed, but was greatly aggravated by the operation. The dentin at the floor of the cavity was probably infiltrated by bacteria, which would account for the migration of leukocytes into the dentinal tubules. These infected dentin splinters, pressed into the already inflamed pulp, caused an acute flareup of the inflammation. The red blood cells in the tissue came from some small vessels that were opened by exposure of the pulp and penetration of dentin splinters. The fibrinous demarcation of several dentin splinters, and the enclosure of a beginning abscess, were also of recent origin, and it is very likely that this occurred during the two-hour period between exposure of the pulp and extraction of the tooth.

The entire pathologic process probably would have run the same course even if the patient had not been exposed to lowered atmospheric pressure. It is, however, possible that the inflammatory reaction and pain would have been somewhat milder.

P265 was an upper first molar in which an old amalgam filling had been replaced about four months earlier. The patient had a sharp pain the night after the filling was placed. In the decompression chamber, he had pain at 5,000 feet. On a second trial run, he again felt pain at 5,000 feet, but, owing to technical difficulties, he was carried to 10,000 feet before being returned to ground level. At the clinic, the patient reported that he had almost fainted because the pain had been so severe. On removal of the amalgam filling, the pulp was found exposed. The tooth was extracted.

Histologic examination revealed an acute inflammation in the pulp horn where the exposure occurred. (Fig. 11*A*.) Large, wide empty spaces were found there, surrounded by leukocytes and red blood corpuscles. (Fig. 11*B*.) Some small pulp stones were present in this area, one of which lay close to one of the bubbles indenting its side.

P245 was a right upper second molar in which an old amalgam filling was replaced two weeks before extraction. There had been no pain before placing the new filling. For one week before extraction, the tooth ached daily when the flight was at 5,000 to 6,000 feet. On the day of extraction, the pilot had a sharp pain at 6,000 feet, then a continued dull ache. There was another sharp pain at 30,000 feet, and again at 25,000 feet on the way down. In the clinic, one hour after descent, a dull ache still persisted. The reaction to ice was almost normal and no pain was felt on percussion.

Histopathologic investigation revealed an abscess with a necrotic empty center and an acute inflammatory reaction in the mesiobuccal area of the pulp chamber. (Fig. 12*A*.) The odontoblasts showed signs of decomposition in some areas, but could be recognized as limiting a layer of predentin around the pulp chamber. Close to the mesiolingual pulp horn, the cavity reached almost into the

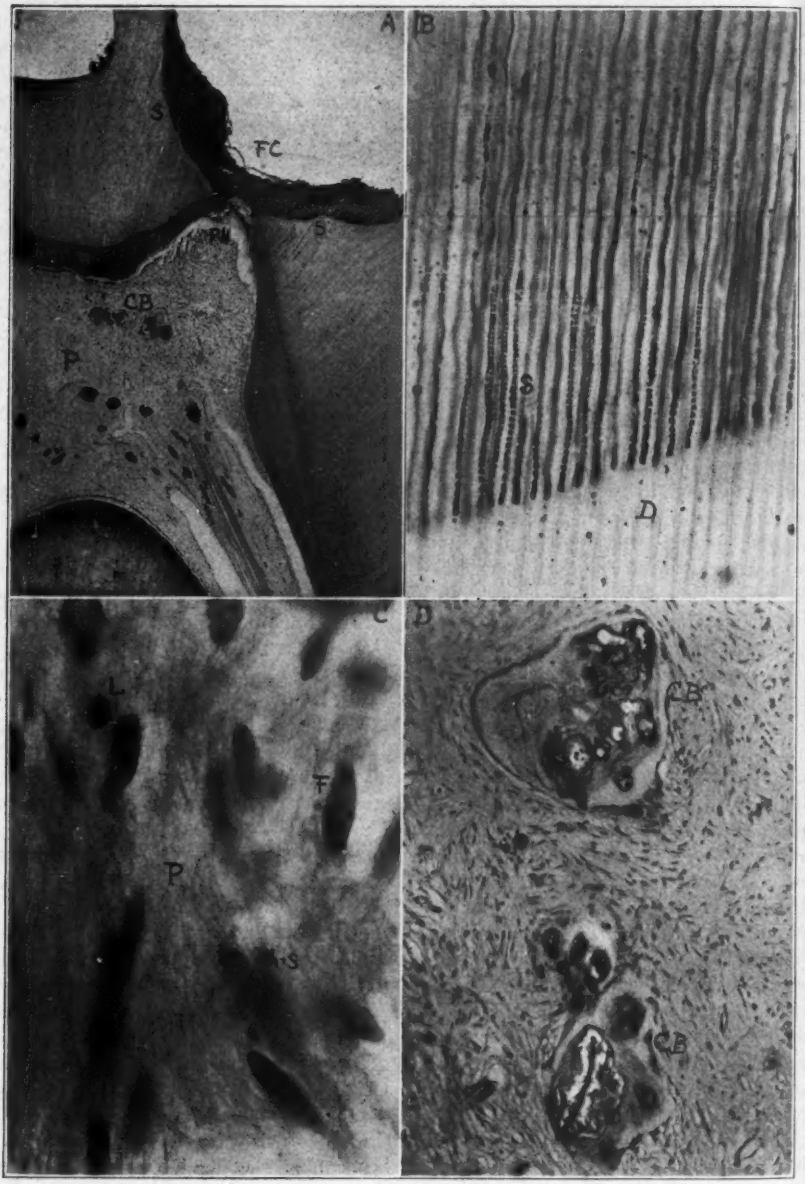

Fig. 15.—P212: Pain at 7,000 feet: *A*: General view showing floor of cavity (*FC*) close to pulp horn (*PH*); *S*, silver precipitate in dentinal tubules; *CB*, calcified bodies in pulp (*P*). *B*: Higher magnification of silver precipitate (*S*) in dentinal tubules; *D*, dentin free from silver. *C*: Higher magnification of pulp tissue (*P*); *L*, lymphocyte; *Ph* + *S*, phagocyte containing silver granules; *F*, fibroblast. *D*: Calcified bodies (*CB*) consisting of calcified necrotic tissue surrounded by newly formed irregular dentin. (Lower second molar.)

pulp. There was only a small amount of irregular dentin formation. Numerous leukocytes surrounded the abscess cavity, and at some points showed denser accumulation. (Fig. 12*B*.) There was a fibrinous network in this area and the meshes between the fibers were filled with leukocytes. In the center, there was necrotic tissue, probably containing bacteria. Around the abscess cavity, the tissue was in a state of progressive necrosis. Many cell remnants could be observed without nuclei. Polymorphonuclear leukocytes and macrophages dominated the picture, exhibiting phagocytic activity. (Fig. 12*C*.) Some of the blood vessels showed signs of thrombosis. (Fig. 12*D*.) There were several small pulp stones in the pulp chamber.

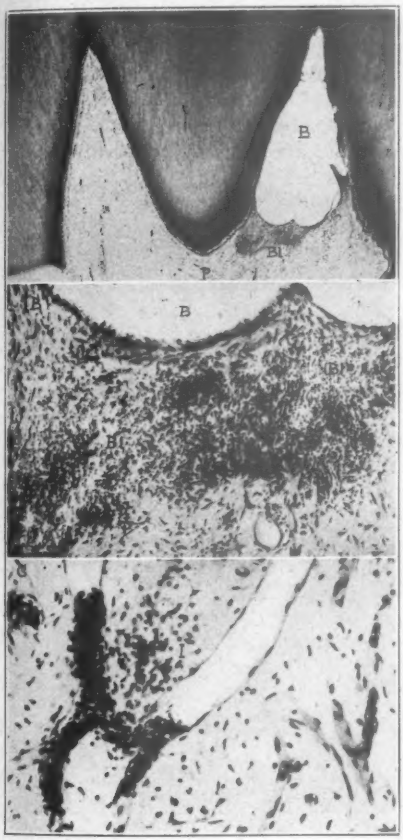

Fig. 16.—P238: Pain at 18,000 feet; intolerable at 23,000 feet: *A*: General view showing bubble (*B*) in pulp horn; bleeding (*Bl*) toward pulp; *P*, pulp. *B*: Higher magnification of area of bleeding (*Bl*); *B*, bubble. *C*: Higher magnification of inflammatory area (*I*) in pulp. The inflammatory cells are lymphocytes. (Upper first bicuspid.)

Correlation of the clinical and histologic findings indicates an acute reaction in the pulp, probably brought about by the operation of replacing the old amalgam filling. There was some slight irregular dentin formation around the pulp chamber, which indicates that there may have been some previous irritation of which this specimen gave no sign. The present inflammatory condition and formation of the abscess probably developed during the two weeks following the dental operation.

Pain in this case started before exposure to decompression, but after the filling was placed. The tooth became sensitive to heat and cold. Low atmospheric pressure brought about the symptom of pain, but, presumably, the tooth would have succumbed under normal conditions, and decompression merely precipitated the onset of pain.

P286 was an upper cuspid with an amalgam filling on the distal side and a deep carious cavity on the mesial side. On excavation of the cavity at the clinic, the patient complained of pain. The pulp was found to be exposed by the carious process. A filling of zinc oxide and eugenol was placed. The pain subsided and, three days later, the tooth was extracted. No decompression test was made.

Histologic investigation revealed a large acute abscess in the pulp chamber, extending over its entire width. (Fig. 13.) Within the abscess cavity, some splinters were seen, which had been pressed into the pulp chamber at the time

of excavation. Apically from the abscess cavity, the dentin was irregular. The rest of the pulp showed a mild degree of inflammation.

In P226, the patient reported at the clinic complaining of pain in the lower left molar area. A defective amalgam filling was found in the second molar. In an experimental run in the decompression chamber, pain started at 11,000 feet, increased at 18,000 feet and persisted up to 30,000 feet, but was relieved at 24,000 feet. At ground level, the patient reported that he felt pain in the area of the upper left lateral incisor, lower left central incisor and lower left second molar. The upper lateral incisor responded very slowly to clinical tests for vitality. An old silicate filling was removed and the pulp found exposed. A drop of pus exuded as if under pressure. In the cervical region, the pulp seemed to be vital. A wire was passed through pulp and apex to insure an opening to the periapical tissue. The patient returned to the decompression chamber and registered no pain in any of the previously sensitive teeth. The same condition was confirmed after extraction of the lateral incisor.

Histopathologic investigation revealed an acute and total pulpitis with abscess formation in the coronal portion of the tooth. (Fig. 14*A*.) There was a dense infiltration of polymorphonuclear leukocytes with a considerable number of red blood corpuscles free in the tissue. The blood vessels were filled with blood throughout the entire pulp. Some vessels were packed to capacity with blood cor-

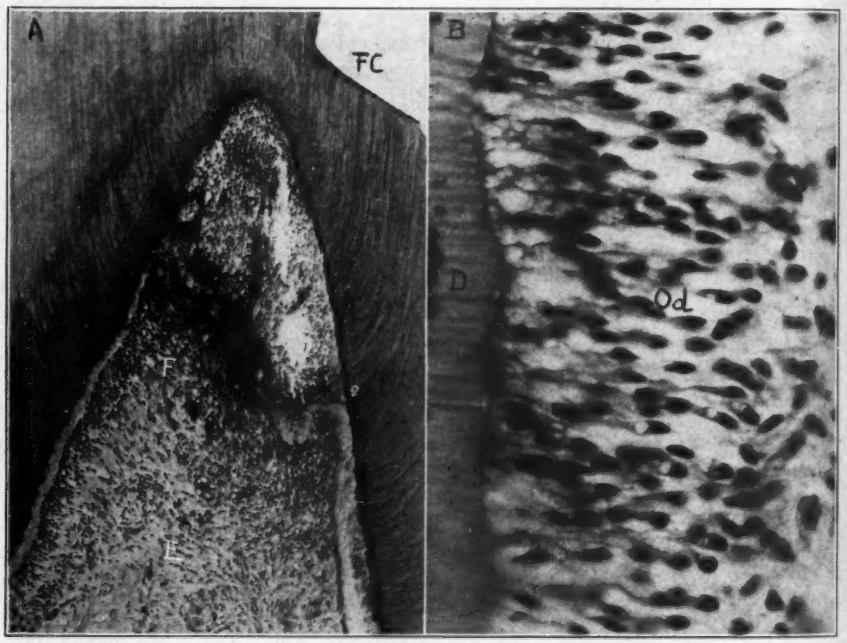

Fig. 17.—P251: Pain at 18,000 feet: *A*: Floor of cavity (*FC*) close to pulp horn (*PH*), which is filled with necrotic tissue and is walled off by a thick layer of fibrinous exudate (*F*). *E*, edematous pulp. *B*: Higher magnification of odontoblasts (*Od*) showing intracellular and extracellular edema; *D*, dentin. (Upper second bicuspid.)

puscles and could be considered in a state of stasis. Circulation had ceased in a large number of vessels. (Fig. 14*B*.) There was considerable edema, and many leukocytes were found in the edematous fluid, as well as between the still-present odontoblasts. In some areas, the odontoblasts were separated from the predentin by edematous fluid and a large number of leukocytes had accumulated in this layer. (Fig. 14*C*.) A fibrinous network had formed in many places, and leukocytes and red blood corpuscles were lodged between the meshwork of fibrin. The acute inflammatory reaction extended to the apical end of the pulp. In the apical region of the pulp canal, a repaired resorption area could be observed. (Fig. 14*D*.) The resorption, presumably, took place a considerable time before the present inflammation since no osteoclasts could be found. Resorption had ceased and was repaired, first by irregular and then by regular dentin. The newly formed dentin was not well calcified.

Correlation between the clinical history and the histologic finding indicates an acute pulpitis in a tooth that had old and extensive silicate cement fillings. From clinical experience, it is known that such sudden and acute inflammatory reactions may, without any apparent exciting cause, occur in teeth containing silicate fillings. Sometimes, such a flareup occurs after an injury or on renewal of a silicate filling and, occasionally, without any apparent provocation. In the present instance, it is conceivable that the acute exacerbation was

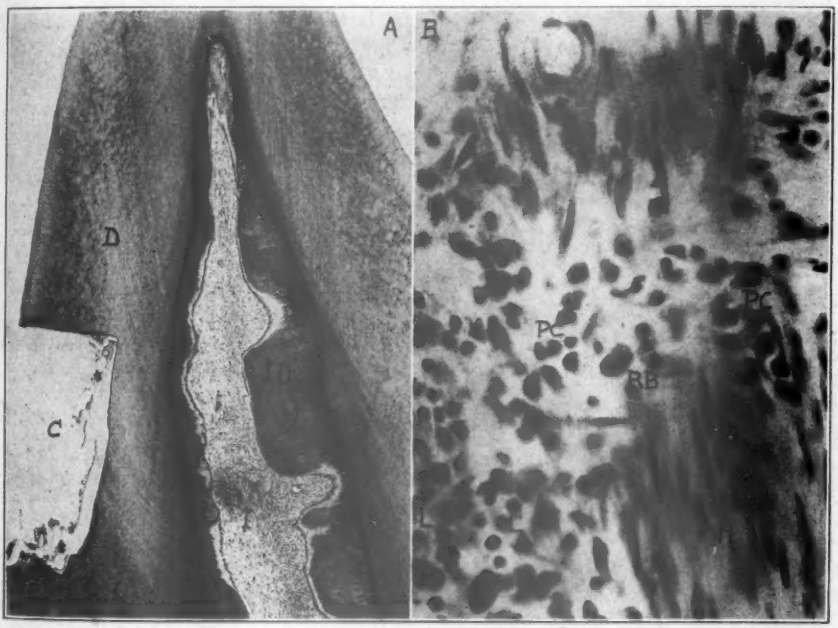

Fig. 18.—P252: Pain at 18,000 feet: *A*: General view showing cavity (*C*) that was filled with silicate; *I*, inflammatory reaction close to nerve bundle; *D*, dentin; *ID*, irregular dentin. *B*: Higher magnification of area *I*; *N*, nerve tissue; *PC*, plasma cells; *RB*, Russell's bodies (degenerating plasma cells); *L*, polymorphonuclear leukocytes. (Upper central incisor.)

brought on by physical irritation or exposure to decompression. However, there evidently was some disturbance in this pulp before the low-pressure incident, as is indicated by the dentin resorption within the root canal. Such resorption might occur under different conditions, especially in chronic irritation associated with a circulatory disturbance.

The foregoing nine cases are characteristic of those cases showing various degrees of acute pulp inflammation. The average altitude for the occurrence of pain for the entire group of seventeen cases was 7,000 feet, the lowest being 2,000 and the highest 18,000 feet.

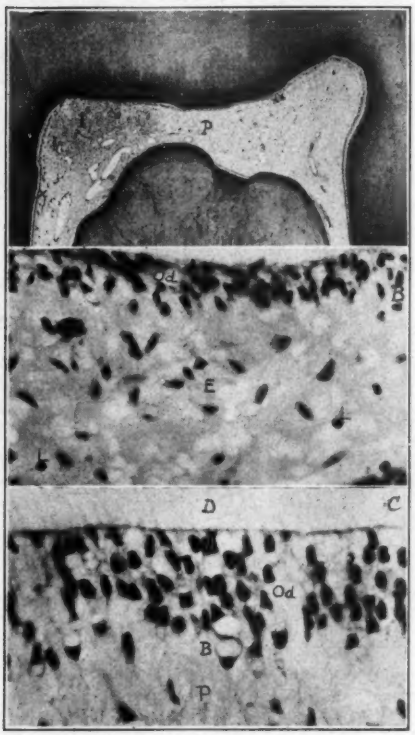

Fig. 19.—P217: Pain at 21,000 feet: *A*: General view showing inflammatory reaction (*I*) in one pulp horn; *P*, pulp. In the inflamed area, blood vessels are congested. Toward the root canal, there are wide blood vessels. *B*: Higher magnification of pulp tissue of area *X* showing edema (*E*); *L*, lymphocytes; *Od*, odontoblasts. *C*: Higher magnification of odontoblasts (*Od*) of area *XX*, showing intracellular and extracellular edema; *B*, ballooning odontoblasts; *P*, pulps; *D*, dentin. (Lower first molar.)

Teeth with Chronic Pulpitis.—The following group of cases was characterized by chronic pulp inflammation. Of the seventy-five cases, fifteen were found to belong to this group, of which nine will be described in detail. As in the previous sections, the cases are presented in the order of severity of the inflammatory process.

P212 was a lower second molar that had been filled about ten weeks earlier. Two weeks later, the patient had some pain in the decompression chamber. Four weeks later, he complained of occasional unprovoked pain and the characteristically persistent sensitivity to cold. Four weeks later, pain forced him down from 7,000 feet. He felt relief immediately on descending. At the clinic, there was no pain to percussion and the reaction to ice was slow. The tooth was extracted one hour after the return to ground level.

The microscopic specimen of the tooth (Fig. 15*A*) showed the cavity close to the pulp horn, reaching almost to the irregular dentin layer which was formed in it. Silver nitrate penetrated through the dentinal tubules, and this silver precipitate was sharply outlined. The silver was deposited in the dentinal tubules as a fine amorphous mass. (Fig. 15*B*.) The deposit was heaviest at the innermost border of silver penetration. Some silver particles passed between the regular and the irregular dentin, and even through the latter into the pulp. Fine particles of silver could be discerned, phagocytosed in some macrophages. (Fig. 15*C*.) The pulp tissue itself showed relatively little pathologic change. There were few lymphocytes and macrophages, and the pulp showed signs of slight edema. There were a considerable number of pulp stones in the pulp chamber and root canal. Close to the pulp horn, some

calcified bodies, other than pulp stones, could be observed, calcified necrotic elements frequently found in chronically inflamed pulps, especially when exposed. The necrotic areas were surrounded by newly formed hard tissue, irregular dentin. (Fig. 15*D*.)

In analyzing the clinical history and microscopic findings of this case, it may be said that the placement of a filling or treatment of the carious cavity with silver nitrate led to a slight, but seemingly persistent irritation, causing some circulatory disturbances; hence, the slight degree of chronic inflammation, especially at the pulp horn closest to the cavity. No changes could be detected that would indicate damage to the pulp from the lowered atmospheric pressure. Any change due to decompression was not of a permanent nature. Probably, the altered circulatory conditions were aggravated by the changes due to low atmospheric pressure, bringing about the symptom of pain.

P238 was an upper first bicuspid. At the clinic, the patient complained of a sore tooth, slightly sensitive to percussion and persistently hypersensitive to ice. There was a large amalgam mesioclusodistal filling in the tooth. In the decompression chamber, the flyer had intolerable pain at 18,000 and 23,000 feet, and asked to be returned to ground level. He remarked that a parachute jump would have been preferable to continued actual flight with such pain. The tooth was extracted one-half hour after the return to ground level.

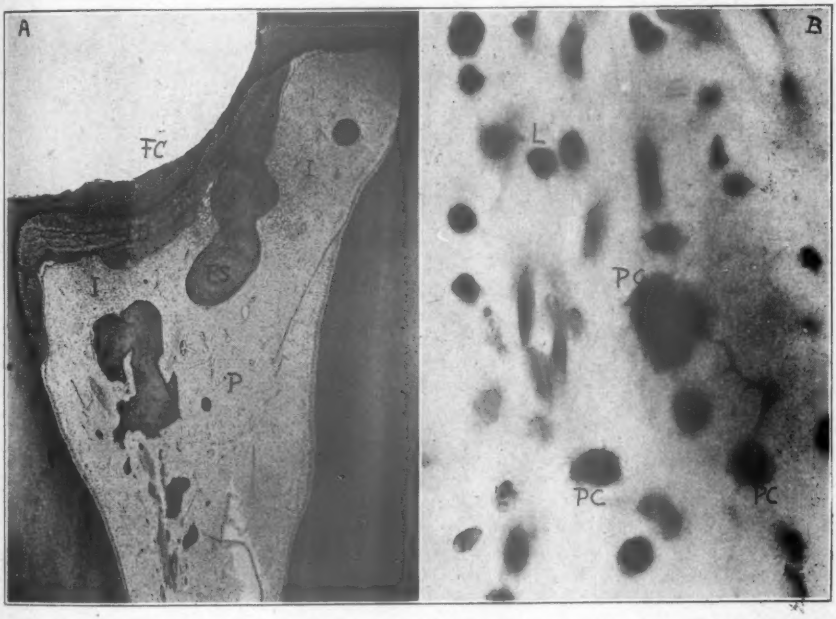

Fig. 20.—P230: Pain at 33,000 feet; *A*: General view showing floor of cavity (*FC*) close to pulp chamber separated from it only by irregular dentin (*ID*); *I*, chronic inflammation in pulp; *P*, pulp; *PS*, pulp stones. *B*: Higher magnification of inflammatory area showing plasma cells (*PC*) and lymphocytes (*L*). (Upper first molar.)

Microscopic investigation (Fig. 16*A*) revealed a mild degree of chronic inflammation in the pulp horn and in re-

stricted areas within the pulp chamber. (Fig. 16*C*.) The most striking feature of this specimen was the large empty space extending over one entire pulp horn. There was a considerable hemorrhage toward the pulp tissue (Fig. 16*B*), whereas the rest of the pulp was free from inflammatory cells, but showed considerable edema. Congested as well as empty and dilated vessels were present.

tion, the tooth showed a normal reaction and extraction was postponed. Ten days later, the tooth seemed to react considerably to cold and the pain lingered. The filling was removed and a slight pulp exposure was detected. The tooth was extracted.

The histologic picture showed necrosis in the pulpal horn below the cavity. (Fig. 17*A*.) The necrotic tissue in the

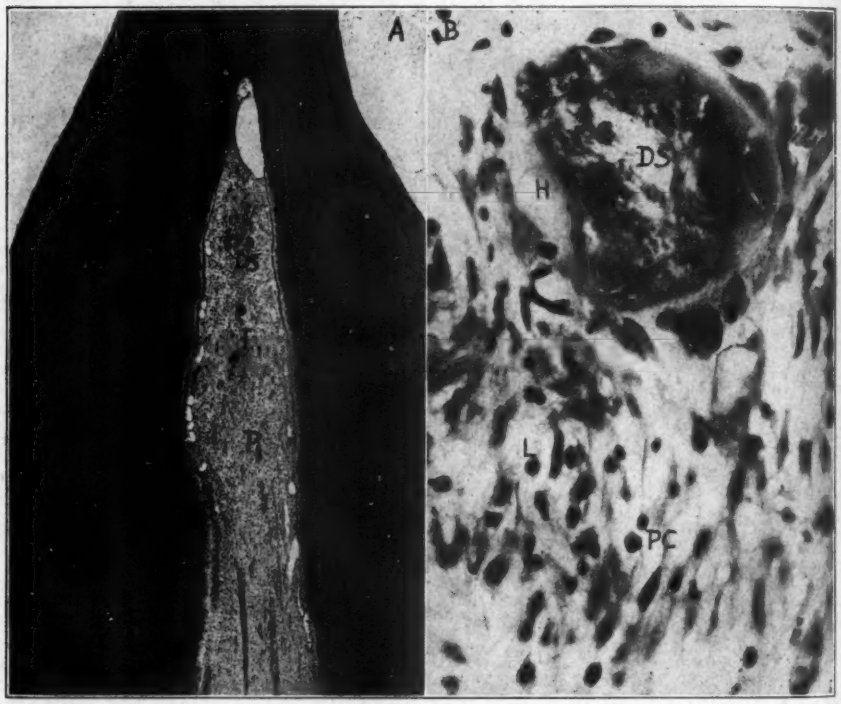

Fig. 21.—P278: Zinc oxide-eugenol filling; no pain: *A*: General view showing chronic inflammation (*I*) in pulp (*P*). Dentin splinters (*DS*) are scattered in the pulp chamber. There are dilated vessels along the odontoblasts. *B*: Higher magnification of area *DS*; dentin splinters (*DS*) surrounded by hyalinized (*H*) and partly calcified tissue; *L*, lymphocytes; *PC*, plasma cells. (Upper central incisor.)

P251 was an upper left second bicuspid. The patient was in the decompression chamber three times and could not rise above 18,000 feet because of pain. There was a new amalgam filling in the bicuspid. On clinical investiga-

pulp horn was walled off from the remaining pulp by a thick barrier of fibrinous exudate. The surrounding pulp tissue was highly edematous and showed disseminated inflammatory cells of the lymphocytic type. The edema extended

into the adjoining odontoblastic layer. (Fig. 17*B*.)

Correlation of these findings indicates that, on exposure of the pulp, a relatively mild reaction set in, localized in the pulp horn that was walled off. It is possible that, under normal conditions, the pulp might have been saved by further blocking off of the damaged area. Exposure to low atmospheric pressure seems to have aggravated the condition and led to increasing sensitivity of the tooth, and thus to its extraction.

P252 was an upper left central incisor containing a silicate filling of recent date. For two weeks, there was no discomfort, but pain set in at 18,000 feet in the decompression chamber. The filling was removed and zinc oxide and eugenol sealed in the cavity. The patient felt comfortable for two weeks. The zinc oxide and eugenol was then replaced by silicate cement. Two days later, during a decompression test, pain occurred at 18,000 feet. The tooth was extracted.

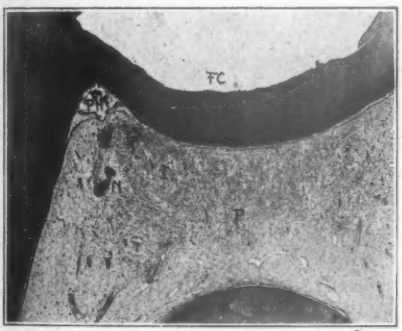

Fig. 22.—P229: No pain at 38,000 feet: *FC*, floor of cavity close to pulp; necrotic pulp horn (*PH*); chronic inflammation (*I*) of entire pulp chamber (*P*); necrotic tissue remnants (*N*). (Lower first molar.)

Histopathologic study (Fig. 18*A*) revealed the accumulation of inflammatory cells in one area of the pulp. There was considerable irregular dentin formation, especially on the lingual side of the pulp chamber. The inflammatory reaction was indicated mainly by lymphocytic elements, plasma cells and macrophages. (Fig. 18*B*.) Some polymorphonuclear leukocytes were disseminated between the nongranular cells of the inflammatory reaction. The inflammatory exudate was in close proximity to a nerve bundle; in fact, many of the inflammatory cells were within the nerve bundle. The inflammation did not extend into the root canal, but was confined to the pulp chamber. There was a slight edema and some small pulp stones were found in the root canal.

Correlation of the clinical and histopathologic investigations explains the pain symptom. There was, probably, considerable irritation in the pulp prior to replacing the silicate filling, and the presence of a considerable amount of irregular dentin strengthens this theory. Furthermore, the presence of plasma cells indicated that the inflammatory reaction was of long standing, some of the plasma cells even showing signs of degeneration in the form of Russell's bodies. There was also an acute reaction, indicated by the presence of polymorphonuclear leukocytes. The chronic inflammation may have been present in the pulp before the first silicate filling was placed, or it might have been due to chemical irritation from the silicate material. The acute inflammation probably started within the last two weeks of the life of the zinc oxide and eugenol filling. Under normal conditions, this tooth might have remained free from pain for some time, but the symptoms were aggravated by the physical impact of low atmospheric pressure. However, even under normal conditions, the pulp would have succumbed to the damage sooner or later.

P217 was a lower first molar with a large amalgam filling. The tooth caused no unprovoked pain until three weeks before extraction. It hurt after one-half hour at 28,000 feet and the pain persisted for three hours after the return

to ground level. On the day of extraction, the pilot was forced down from 21,000 feet, but was relieved from pain at 9,000 feet. The tooth was extracted about one hour after descent.

Microscopic examination revealed a chronic inflammatory reaction in the pulp horn closest to the cavity. (Fig. 19*A*.) There was a congestion of the blood vessels in this area and most of the vessels were filled to capacity with blood. Apically from the congested area, numerous empty but distended blood vessels could be seen. The tissue was densely infiltrated by lymphocytic elements and macrophages. There were no polymorphonuclear leukocytes to be found, this fact indicating the chronic nature of the inflammatory reaction, which was due to an irritation of long standing. The odontoblasts in the pulp horn had degenerated in some restricted areas. There were numerous wide, empty veins adjoining this part of the pulp. Toward the root canal, and especially close to the odontoblastic layer, some of the smaller vessels were filled with blood. The pulp in this region showed a considerable degree of edema. (Fig. 19*B*.) The cells of the pulp were separated from one an-

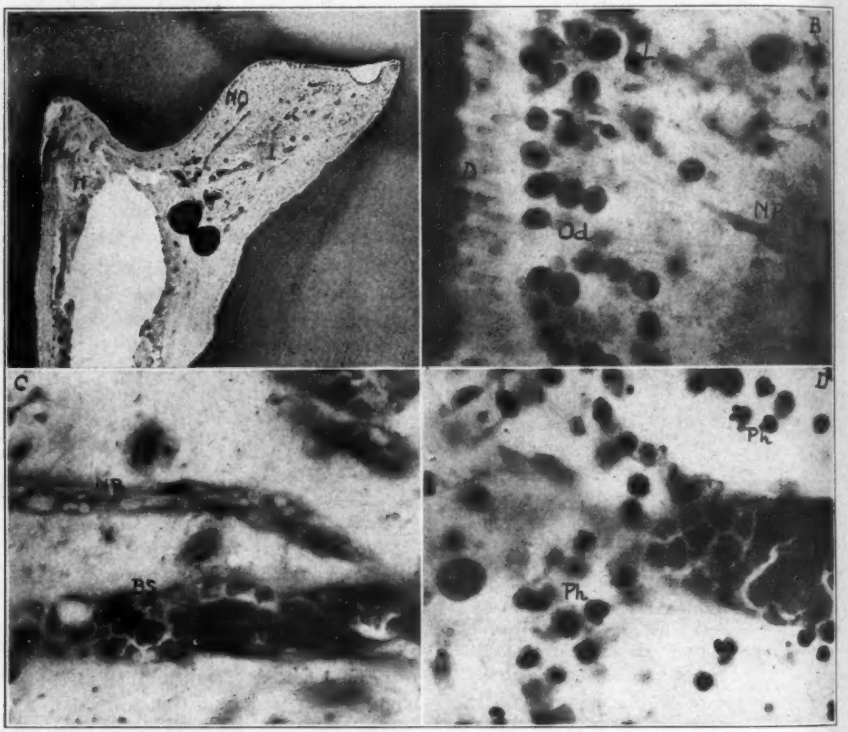

Fig. 23.—P231: Pain on descent: *A*: General view showing necrotic area (*N*) in pulp chamber; no necrotic odontoblasts; *I*, inflammatory reaction. *B*: Higher magnification of necrotic odontoblasts (*Od*). The pulp tissue is also necrotic (*NP*). *L*, lymphocytes; *D*, dentin. *C*: Necrotic blood vessel (*NB*); stasis in blood vessel (*BS*). *D*: Higher magnification of inflammatory area showing phagocytes (*Ph*). (Upper first molar.)

other by tissue fluid in which round spaces could be seen, caused by shrinkage of coagulated edema fluid. There were few lymphocytes in the edematous tissue. Edema in the pulp was found not only close to the pulp horn, showing the chronic inflammatory reaction, but also elsewhere in the pulp chamber. The odontoblasts showed clearly the effects of edema. (Fig. 19*C*.) In the cytoplasm of the odontoblasts, edema fluid appeared, and some of the cells were blown up like balloons. Edema appeared in the odontoblasts on each side of the nuclei and also between the cells.

In analyzing the clinical picture of P217 and the microscopic findings, it

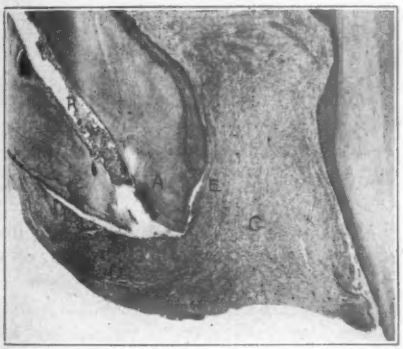

Fig. 24.—P241: No pain at 38,000 feet. Apical region of one root (*R*) of molar; *A*, apex. The root canal is filled with leukocytes and necrotic tissue. *G*, granulation tissue; *E*, epithelium; epithelial granuloma. (Upper first molar.)

can be stated that the pulp of the tooth was in a condition of chronic inflammation, due to some irritation extending from the cavity. The congestion of the blood vessels and the accumulation of lymphocytic elements were signs of this prolonged irritation. The extent of the edema, with the ballooning cytoplasm of the odontoblasts, might have been due to low atmospheric pressure. The edema was probably due to a transudate and not to an exudate since the cellular elements of an inflammatory exudate were absent in most parts of the pulp. The inflammatory exudate was confined to one pulp horn. The increased filtration of fluid from the vessels was probably made possible by increased capillary pressure and by the change in the permeability of the vessels. The patient was subjected to an altitude of 21,000 feet, and the tooth was extracted one hour after descent. It is possible that, within this hour, owing to the altered circulatory conditions, the edema could not be overcome by resorption of fluids into the veins and lymph vessels. In teeth extracted under normal conditions, edema seldom is of this extent. This is especially true of edema of the odontoblasts, where it is frequently observed. In the 210 control cases, a similar degree of ballooning could not be found. It can be concluded that there was irritation and inflammation in this tooth before it was exposed to low atmospheric pressure, and that the circulatory disturbances, together with the mechanical effect of decompression, led to development of the pain symptom. That the pain was observed three weeks before extraction, at an altitude of 28,000 feet, was *per se* an indication of circulatory difficulties in this tooth. It is also possible that the large number of empty wide veins was due to escape of nitrogen from the blood accumulated in these veins (nitrogen emboli), causing congestion in the peripheral vessels drained by the veins.

P230 was an upper right first molar, with a deep cavity and amalgam filling. In the decompression chamber, the patient had one sharp pain at 33,000 feet, then a dull ache up to 38,000 feet. Pain subsided after a few minutes at 38,000 feet and did not recur during descent. One-half hour after descent, at the time of extraction, the tooth was not sensitive to percussion or ice.

Histopathologic investigation revealed considerable irregular dentin formation below the floor of the cavity (Fig. 20*A*),

laid down in several layers. Calcification was incomplete, as indicated by the numerous interglobular spaces. At one point, the irregular dentin came in contact with a pulp stone with the result that the free pulp stone became attached to the roof of the pulp chamber. There were many other pulp stones in the pulp chamber and root canal, where considerable hyalin degeneration and calcification could be observed. The calcification was especially marked along the connective tissue surrounding the nerve bundles and vessels. In the pulp chamber, below the cavity, an inflammatory infiltration could be seen. The cellular elements of this inflammatory reaction were lymphocytic elements, plasma cells and macrophages. (Fig. 20*B*.) Only a few polymorphonuclear leukocytes were close to the irregular dentin between the pulp stones and roof of the pulp chamber, but none were seen deeper in the pulp. There was a slight edema in the entire pulp chamber.

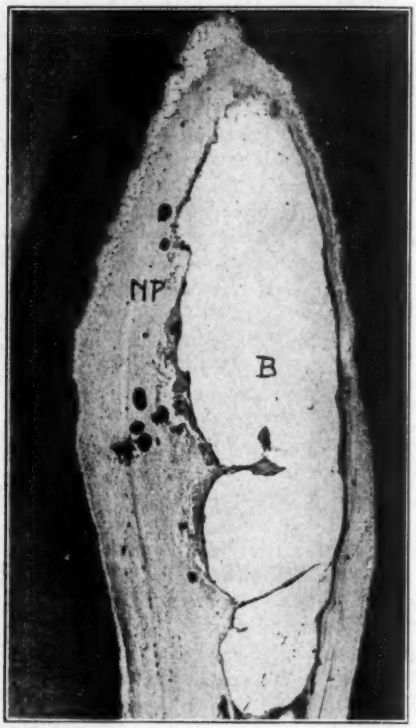

Fig. 25.—P275: Necrotic pulp (*NP*) with wide empty space (*B*), probably blood vessel. (Upper cuspid.)

The pain in this case is explained by the chronic inflammatory reaction and circulatory difficulties, combined with the mechanical effects of decompression. These circulatory difficulties were mainly expressed by the hyalin degeneration and extensive calcification in the pulp chamber and root canals. Irritation in the pulp chamber, which was of long standing, can be diagnosed from the considerable amount of irregular dentin. Despite apparent circulatory difficulties, an increased pressure in the pulp was probably equalized; hence, the relatively rapid relief from pain. Clinical investigation indicated a relatively low degree of sensitivity (no pain on application of ice) in this particular case.

P278 was an upper right central incisor. At the clinic, the patient complained of sensitivity to sweets and cold. The tooth had a defective silicate filling. There was no pain on percussion and no unprovoked pain, but great sensitivity to ice. The silicate filling was removed and a zinc oxide and eugenol filling was placed. The patient went to 38,000 feet in the decompression chamber without pain. The following day, the zinc oxide and eugenol filling was removed and a small drop of blood oozed out of the pulp horn. The tooth was then extracted.

The histologic specimen revealed a chronic inflammation in the entire coronal portion of the pulp. (Fig. 21*A*.) Several small dentin splinters could be seen, embedded in the tissue and enclosed in newly formed calcified matrix. (Fig. 21*B*.) This indicates that the pulp was exposed at the time of placing the silicate filling. Many blood vessels along the odontoblastic layer were empty and

dilated, but, in the center of the pulp, the vessels were filled with plasma and blood corpuscles. Toward the root canal, the blood vessels seemed to be highly congested. In the apical portion, the pulp tissue showed hyalin degeneration and calcific deposits.

P229 was a lower first molar. The tooth was free from symptoms, but was marked for extraction because of a pulp exposure from caries, which was disclosed during excavation. The patient made a painless ascent to 38,000 feet.

The histologic specimen (Fig. 22) showed a chronic inflammation extending into the entire pulp chamber, but most severe at the pulp horn where the exposure occurred. In the tip of the pulp horn, some necrotic tissue was seen partly separated from the pulp by a layer of uncalcified irregular dentin, which was seemingly formed after the exposure occurred. Calcified necrotic cell remnants could be observed in several groups. No irregular dentin had been formed and there were no odontoblasts at the roof of the pulp chamber. At its side walls, they were present. There was a mild edema.

P231 was a right upper first molar. The patient came to the clinic complaining of pain in this area. The tooth was sensitive to percussion, but showed no sensation on application of ice. In the low pressure chamber, the pain decreased on the climb to 38,000 feet and increased during descent. The tooth was extracted one-half hour after descent. On removal of the amalgam filling and grinding of the tooth for histologic fixation, the pulp was exposed and a fetid odor characteristic of gangrenous pulps was noted.

Histologic investigation revealed a partly necrotic pulp. In some parts, the last attempts at tissue defense were still discernible. Some of the necrotic tissue was lost by complete decomposition and an abscess cavity had formed, surrounded by partly necrotic tissue. (Fig. 23*A*.) The odontoblasts showed karyorrhexis and karyolysis. (Fig. 23*B*.) The cells were rounded and the nuclei were completely missing in some of the cells, others being on the way to complete disintegration. A few lymphocytes could be seen close to the odontoblasts as well as deeper in the pulp. Some of the blood vessels showed stasis, others more or less complete necrosis. (Fig. 23*C*.)

A large number of macrophages were gathered where the necrosis was incomplete. (Fig. 23D.) In some of these cells, necrotic cell remnants were seen, signifying active phagocytosis. Some of the phagocytes also showed signs of beginning necrosis and dissolution. This case is exceptional because the pain occurred at ground level and decreased during ascent, increasing again on descent. Similar incidents of pain are reported in maxillary sinus complications and also in disturbances of the middle ear.

The foregoing cases with chronic pulp inflammation are characteristic for the entire group. Pain occurrence level in the fifteen cases in this group varied between 5,000 and 36,000 feet, the average being 17,000 feet.

Teeth with Nonvital Pulps.—The histologic material included seven teeth with nonvital pulps. P239 was an upper left lateral incisor with an incomplete root canal filling. Roentgenographically, there was evidence of periapical bone rarefaction. Clinically, the tooth was free from painful symptoms and was painless in the decompression chamber up to 38,000 feet. Microscopic investigation revealed an empty pulp chamber and a canal showing only some tissue detritus. In the apical portion of the root canal, some fibrous tissue could be seen infiltrated with lymphocytic elements. Some periapical tissue which remained attached to the apex after extraction also gave evidence of the presence of chronic inflammatory elements.

P241 was an upper left first molar with a nonvital gangrenous pulp. The

patient developed a painless swelling during the night and was sent into the decompression chamber next morning. He had no pain or additional swelling up to 38,000 feet. Microscopically, the pulp was completely decomposed and, in the apical area, there was an area of acute inflammatory reaction superimposed upon a chronic epithelial granuloma. (Fig. 24.)

Some of the other teeth with nonvital pulps developed a slight pain sensation after two hours at a 38,000 foot level.

P275, an upper left cuspid, was of special interest because it had been observed for some time before extraction. For six months, the patient made three trips to 38,000 feet each week in the decompression chamber. The tooth was filled with silicate cement several months before extraction. The tooth was very sensitive at the time of filling. Three weeks afterwards, it was painful for a brief time at 18,000 feet. Similar pains occurred twice the following week. No further pain was felt during the next three months. Then the patient had some unprovoked pains during the night and developed a swelling on the following day. The tooth was extracted. Histologically, the pulp in this tooth consisted of necrotic tissue of homogeneous appearance (Fig. 25), the nuclei staining only faintly. There was a large empty space in the coronal portion as well as in the root canal. The outline of these spaces suggested extremely expanded veins.

COMMENT

The mechanism by which high altitude flying causes toothache apparently can be explained only on the basis of the general physical changes that occur in the body under decompression; namely, liberation and expansion of gases from the blood and tissue fluids. In themselves, these changes do not elicit pain in teeth with normal pulps. The predisposing pathologic process in all cases of toothache in high altitudes is a circulatory disturbance in the pulp that prevents equalization of pressures. A pulp enclosed in unyielding dentinal walls is always a *punctum minoris resistentiae* to disturbances of circulation because of the vicious circle instigated by an increase in intradental pressure.

Clinical observation as well as histopathologic investigation supports the generally accepted contention that decompression does not cause pain in the teeth with normal pulps. One of the three teeth with normal pulps that were investigated did not show any changes that could have been attributed to lowered barometric pressure, even at 3 pounds per square inch, which is about one-fifth of the atmospheric pressure at sea level. One of the cases (P228) showed a definite change, probably due to lowered atmospheric pressure, the presence of empty dilated blood vessels. The blood of these vessels had been pressed into the smaller branches and vessels nearer the periphery, which were well filled and congested. There is a possibility that a certain degree of irritation was already present at this point owing to the proximity of the cavity to the pulp horn. The lowered atmospheric pressure might then have aggravated the condition. It is probable that the "empty" dilated blood vessels are filled with nitrogen released during decompression. The absence of any pain symptoms in teeth with normal pulps proves that the increase in pressure from nitrogen bubbles is within the limits of the regulatory capacity of the circulation of a normal pulp. Wherever this circulation is impaired, the added impact of nitrogen bubbles will throw the circulation in the pulp into disorder, and the circulation cannot be regulated in time to prevent pain. In edema of the pulp and acute or chronic pulpitis, there is, therefore, a predisposition to high altitude toothache.

In sixteen cases, edema was the pre-

dominant histopathologic finding. Pain occurred in these cases at between 2,000 and 25,000 feet, with an average of 11,000 feet. At this time, we are not able to explain the mode of development of this edema, but it seems obvious that there is a correlation between the edema and the pain. It is peculiar that an edema, which is most frequently one of the early signs of inflammation, should exist for any length of time without the other symptoms of inflammation, such as the accumulation of inflammatory cells. One possible explanation for the edema is that, owing to some chemical irritation (amalgam filling), the blood vessels in the pulp have been damaged and thus capillary filtration has been increased. The condition might have been further aggravated by decompression, to a point where pain would set in owing to increase in pressure in the pulp chamber. This increase might be further heightened by the presence of pulp stones, calcification and hyalin degeneration in the root canals, disturbing the circulation and rendering resorption of the increased fluid difficult. This problem is being investigated and results will be reported.

Correlation of clinical observations and microscopic findings in seventeen cases with acute pulpitis revealed that pain was elicited at low altitudes, between 2,000 and 18,000 feet (average 7,000 feet). We can explain this by the fact that, in acute inflammation, the sensitivity of the nerve elements is increased (presence of histamine?). Most of these teeth were painless at ground level and hypersensitive only to ice. Pain might be due to an increase in fluid filtration and liberation and expansion of tissue gases, especially nitrogen, and thus increased pressure within the pulp chamber. That the severity of the pain and the level of its onset depend on the extent of the circulatory changes in the pulp seems plausible.

Pain in the teeth with chronic pulp inflammation occurred at between 5,000 and 36,000 feet, the average for the sixteen cases observed being 17,000 feet. This pain level, which is considerably higher than in the edematous or acute pulpitis cases, seems to be consistent with the relatively lesser sensitivity of chronically inflamed tissue. There are less edema and hyperemia in these cases and, clinically, response to ice application is less intense and briefer.

From a large number of cases of teeth with nonvital pulps tested in the decompression chamber, seven were sectioned for microscopic examination. It can be stated that teeth with nonvital pulps are not commonly a source of pain during decompression. When they are, the pain is dull and persistent, and is sometimes followed by a phlegmonous swelling. Pain tends to develop in these cases after prolonged exposure to very high altitudes or after a flight. Apparently, only those teeth that are on the verge of an exacerbation respond painfully to decompression. This is true for teeth with nonvital pulps, with or without periapical involvement.

In correlating the histologic findings with the clinical observations, large empty bubble-like spaces were observed in several teeth of the groups with edema and acute or chronic pulpitis. Such cases were P258 and P223 in the edematous group; P213, P260 and P265 in the acute, and P238 in the chronic pulpitis group. In checking the history of these teeth, it became apparent that all were the source of severe pain. P258 is shown in Figure 4. Pain was encountered at 2,000 feet and continued to increase until it became intolerable at 10,000 feet. In P223 (Fig. 5), pain set in at 5,000 feet, becoming intolerable at 10,000 feet. In both cases, the principal pathologic condition was edema. P213 (Fig. 7) was so painful that the pilot in actual flight had to dive from 12,000 feet. P260 (Fig. 9) was painful at 5,000 feet, the pain becoming intolerable at

6,000 feet. P265 (Fig. 11) developed pain at 5,000 feet, becoming extreme at 10,000 feet. These three cases belong to the group of teeth with acute pulpitis. One case of the chronic pulpitis group, P238 (Fig. 16), was painful at 18,000 feet and, at 23,000 feet, the flyer asked to be returned to ground level because of intolerable pain.

In analyzing these cases, it can be assumed that gas formed in these pulps. It is possible that, in connection with the inflammatory process, especially in areas where tissue decomposition took place, some gas might have formed even before exposure to decompression, and decompression only aggravated the already existing condition. However, most of these cases do not represent far advanced inflammatory conditions, and we have to take into consideration that the bubble formation may be the result of nitrogen liberation due to decompression. Nitrogen bubbles are not considered a common finding below a level of 18,000 feet, where the pressure sinks to half an atmosphere. However, it must be taken into account that we are not dealing with healthy, but diseased tissue. Bubble formation, except in P260, always took place in the pulp horn where pulp injury was most apparent. It is conceivable that nitrogen gas is liberated at a much lower level in pathologic tissues than in normal tissues. The extreme pain in these cases is probably brought about by the sudden gas expansion and pressure upon the adjacent tissues. The tissues often appear compressed and acute hemorrhage is frequently found. This hemorrhage seems to indicate that the bubble is of very recent origin, corresponding in time to the pain sensation.

Sometimes, similar empty spaces can be observed in teeth with highly inflamed pulps that have not been exposed to decompression. There is no acute hemorrhage in such cases. Bubbles may be found, especially in cases of acute abscess or in teeth in which the pulps have undergone gangrenous decomposition. Empty and sometimes even dilated vessels can be found in pulp specimens that are not well fixed, and where gas formation might have been the result of postmortem blood decomposition or putrefaction. It is believed by some that air may be sucked into the pulp canal during extraction. The presence of bubbles or dilated blood vessels has its diagnostic value only in well-fixed specimens and in combination with other clinical and histologic findings.

In the case of P260 (Fig. 9), the bubbles were not in the pulp horn, but in the root canal below the floor of the pulp chamber. There was a wide empty vessel in this area and, close to it, a large number of small bubbles. The case differs from the others not only in its histology, but also by the fact that this tooth was the only one extracted in the decompression chamber at an altitude of pain development; i.e. 6,000 feet. It would be of especial value to the entire decompression problem if similar experiments were carried out, since the dental pulp is seemingly the only human material readily available for biopsy.

Special attention is focused on P231 (Fig. 23) because it exhibited pain at ground level and the pain decreased during ascent, increasing again on descent. Similar pain occurrence is reported in maxillary sinus complications and disturbances of the middle ear. When the outer atmospheric pressure is lowered, air from the sinus or the middle ear can escape through the openings of the sinus or eustachian tube; therefore, pain rarely occurs in these organs during ascent. However, in descent, air may be prevented from entering the sinus or middle ear by the swollen mucous membranes in upper respiratory infections, this preventing equalization of pressure. The only explanation for this case seems to be that the pulp canal may have been in communication with

the maxillary sinus. In this event, gases in this necrotic pulp tissue could escape through the apex into the sinus, and from there to the outside. With increase in barometric pressure during descent, the entrance of gas into the sinus may have been prevented, thus causing pain.

It should be emphasized that teeth with extensive inflammatory reaction in the pulp are not a source of pain if the cavity is open, as exemplified in P229 (Fig. 22). This is not an observation on an isolated case, but was confirmed repeatedly. Closing the cavity with amalgam, zinc phosphate cement or even with a thick layer of cavity varnish invariably results in pain during decompression. The only feasible explanation for this observation is that sufficient fluids or gases can escape through the dentinal tubules to prevent pain during decompression.

That teeth with silicate fillings seldom, if ever, are a source of pain during decompression, even if they show pulp inflammation, is an unexplained clinical observation as yet.

It has been observed that a tooth with a vital pulp that has been painful during decompression can be rendered painless by a zinc oxide and eugenol cement base or filling. This procedure is advocated by virtually all writers on this subject. Two cases of zinc oxide and eugenol medication are included in this presentation, P286 (Fig. 13) and P278 (Fig. 21). In P286, a toothache at ground level was relieved by filling with zinc oxide and eugenol. The pulp was exposed and, histologically, showed an acute abscess. Nevertheless, the tooth had remained free from pain for three days. P278 presented a chronic pulp inflammation, following exposure. Temporary filling with zinc oxide and eugenol permitted a painless ascent to 38,000 feet. In view of the histopathologic findings as correlated with the clinical observations, it is doubtful whether the use of zinc oxide and eugenol in the case of flying personnel is advisable. There is no doubt that this treatment will relieve pain in decompression, but this does not mean that the pulps are cured or will remain painless indefinitely. No reliable experimental evidence has been offered that the pulp inflammation subsided under the influence of filling with zinc oxide and eugenol. It is more likely that teeth made comfortable for the moment by this medication will become a menace at a later date. With flying personnel, this should be taken into consideration and such medication should be used only as an emergency measure. At the present time, it is not known what effect a zinc oxide and eugenol filling has on a healthy pulp or what renders it a pain-relieving agent.

The diagnosis of a decompression toothache cannot be made by any single clinical test. With care, various bits of evidence will gradually accumulate to a point where a proper diagnosis can be expected. The following procedures provided the diagnostic data on which recommendations for the extraction of these teeth were made.

Case History.—The case history should include the following questions concerning the patient prior to the decompression experience:

1. Previous unprovoked pain, especially at night.
2. Previous hypersensitivity to heat, cold, sweets or pressure.
3. Approximate date of fillings in suspected area.
4. Unusual pain during the filling, or during the following night.
5. Pain during ascent or descent. If on descent, it should include the length of time spent at 38,000 feet.
6. Altitude at which pain occurred. If it occurred at 38,000 feet, it should include the length of time the patient remained at this level.
7. Character of pain: sharp or dull, single or intermittent, intense or mild.
8. Altitude at which pain was relieved.
9. If pain persisted after reaching ground

level, the length of time it persisted and whether it was followed by swelling.

10. Tooth suspected by the patient.

Roentgenograms of the suspected area or side should be available.

Clinical Examination.—This should include:

1. An estimate of the age of fillings in suspected area.
2. Percussion test from the lingual and buccal aspect as well as vertically.
3. Exploration for unfilled cavities or defective fillings.
4. Irritation with ice, heat and electricity.
5. Removal of fillings and excavation of cavities.
6. Repeated decompression tests, if necessary.

An examination of this character should give the following information:

1. Unprovoked pain, especially at night, would be a strong indication of pulp involvement, probably of an acute type.

2. A tooth that has shown previous hypersensitivity should be carefully examined. Sensitivity to heat and not to cold, or relief of pain by cold, would suggest a tooth with a nonvital pulp. Equal response to heat and cold indicates hypersensitivity. Prolonged pain from cold points to pulp inflammation. Hypersensitivity to sweets tends to suggest caries or a defective filling. Pain on pressure indicates periapical irritation, but not necessarily abscess formation. (Occlusal trauma should be excluded.)

3. Amalgam fillings of recent date are more to be suspected than are old ones or other types of fillings.

4. Unusual pain during filling or during the night after the tooth was filled suggests pulp exposure.

5. Whether the pain was encountered during ascent or descent in the decompression chamber would differentiate between teeth with vital and teeth with nonvital pulps. Pain during ascent indicates a tooth with a vital pulp; pain during descent, a tooth with a nonvital pulp, except that there appears to be a "lag" between the irritation and the response and thus a vital tooth might hurt briefly during descent if a very short time had been spent at the maximum altitude.

6. The altitude at which pain occurs during ascent should give some indication as to the type of inflammatory process in the pulp. The lower the pain level, the more acute the inflammation.

7. Pain at 38,000 feet, and particularly after some time at this level, should focus attention on a tooth with a nonvital pulp.

8. Sharp pain during ascent would indicate a tooth with a vital pulp and with a rather acute inflammation; whereas, dull pain during ascent would point to a chronically inflamed pulp. Dull pain during descent would indicate a tooth with a nonvital pulp or an inflamed maxillary sinus.

9. Very intense pain is sometimes associated with bubble formation, even in case of a mild pulp reaction.

10. The sooner the pain is relieved during descent, the more acute the inflammation is apt to be.

11. Pain that persists for a long period after ground level is reached is sometimes associated with teeth with necrotic or nonvital pulps. In cases of necrotic pulp, a phlegmonous swelling may develop within twenty-four hours.

12. Usually, the patient has no difficulty in pointing out the quadrant in which the pain occurred, but his selection of the tooth within the quadrant has been found to be unreliable. It is not unusual to have the pain referred to the region of another tooth, especially the lower bicuspids, even when maxillary teeth are involved.

13. Roentgenograms are important in diagnosis, but they cannot be expected to disclose pulp changes or minute exposures of the pulp.

14. Observation should indicate the approximate age of fillings. Amalgam fillings of recent date can usually be recognized.

15. Percussion and pressure tests are indicative of periapical irritation, but even considerable sensitiveness is no indication for extraction.

16. Unfilled cavities and defective fillings should be explored. The more open a cavity and the more defective the margins of a filling, the less should a tooth be suspected of having been painful during decompression.

17. The best diagnostic aid has been the

response to stimulation with ice. Nearly all teeth with vital pulps will respond to this test, but it is necessary to differentiate between hypersensitive teeth and those reacting with prolonged pain to irritation with ice. In hypersensitive cases, the response is immediate, but recovery is very rapid when the ice is removed. The expression "prolonged pain" signifies slow recovery and lingering pain. The initial response to the ice may be rapid or slow. This test should always be carried out by comparing adjacent and corresponding teeth. Only because of the relative difference in reaction is this useful. Failure of one tooth to respond to ice should suggest a nonvital pulp. It has been frequently observed that patients attempt to cover teeth with the tips of their tongues in the case of prolonged pain from ice. (Ice or ethylchloride on cotton can be used.)

18. Extreme sensitiveness to application of heat would indicate pulp degeneration. Although no actual heat tests were made on teeth showing large bubbles in the pulp horns, it seems reasonable to believe that they would have reacted painfully to heat.

19. Facilities were not available for the electric pulp test in this series of cases, but its use may furnish additional information.

20. The final clinical test is the removal of suspected fillings and excavation of cavities, with particular attention to exposed pulp horns. Exposures do not always show on inspection, but use of a sharp explorer, with some pressure, will usually disclose the concealed exposure area. Staining the cavity with an aqueous iodine solution or with silver nitrate will often mark the exposed area as a small unstained spot. All caries must be removed.

21. Repeated decompression tests will confirm a doubtful diagnosis.

The foregoing outline of diagnostic procedure may seem unduly lengthy in consideration of the fact that the aim of many clinicians is merely to locate the tooth that is painful and not to diagnose the specific nature of the pathologic process in the pulp. It seems of little clinical significance, from the point of view of present-day therapy, whether the pulpitis is acute or chronic, or whether a tooth has bubbles in a pulp horn or not; but, the clearer the picture, in the clinician's mind, of the histologic changes in the tooth, the better the clinical interpretation, especially in doubtful cases. Histologic investigation has indicated that any tooth that has been painful during decompression has a diseased pulp, and since a diagnosis of pulpitis, at the present time, means either root-canal therapy or extraction, such a diagnosis will not be readily accepted by a conscientious operator.

In correlating the etiology of the dental problem with the medical aspects of aviation, there has been nothing in these findings to indicate any possible dental disturbance connected with vertigo. As virtually all cases in this study came from a decompression chamber operated at ground-level temperatures, no correlation with cold could be made. Nothing has been found to indicate any connection between pain of dental origin and acute or chronic air sickness. Toothache occurred during anoxia tests and with the use of oxygen masks. Since the tests have not been controlled in this respect, the possibility of an effect of anoxia on the dental pulp cannot be entirely ruled out, but there are no findings that would indicate anoxia as pain-producing. Controlled experiments in this respect would be desirable. Toothache seems to be mainly associated with the mechanical effects of decompression, including an apparent increase of capillary filtration, dilation of blood vessels, possible nitrogen embolism and bubble formation in the tissues, none of which appear to cause any change in normal tissues.

SUMMARY AND CONCLUSIONS

1. Two hundred and fifty cases of toothache of decompression origin were clinically observed. In seventy-five of these, sections were made for histologic study. Two hundred and ten teeth were used as controls.

2. Of the seventy-five teeth, sixteen

had edematous pulps, seventeen showed acute pulpitis and fifteen chronic pulpitis and seven were teeth with nonvital pulps. In the twenty cases remaining, three involved teeth with normal pulps. The rest belonged in one or the other of the foregoing groups, but could not be used for statistical purposes.

3. Teeth with normal pulps will not hurt under decompression, whether the tooth is intact, carious or filled.

4. A tooth with an open cavity will not hurt during decompression, even if the pulp is diseased. Filling such a tooth with an amalgam or oxyphosphate cement, or lining the cavity with a cavity varnish, will cause pain in decompression. No pain was attributed to teeth with defective fillings or to air bubbles under fillings.

5. Zinc oxide and eugenol will prevent toothache under decompression, but its use is advisable for flying personnel only as an emergency measure, because of possible subsequent complications.

6. Pain in teeth with vital pulps occurs almost invariably during ascent. The average pain experience in edematous cases was at 11,000 feet; in acute pulpitis, 7,000 feet, and in chronic pulpitis, 17,000 feet.

7. Teeth with nonvital pulps will exhibit pain only exceptionally and, in these cases, pain will usually occur during descent.

8. Sudden sharp pain at relatively low altitudes indicates bubble formation in the pathologically changed pulp.

9. The utmost care in examination is necessary because it is difficult not only to diagnose the exact nature of pulp disease, but often also to diagnose the condition of the tooth involved. A diagnostic outline is found useful. Stimulation with ice was found to be the most reliable single clinical test.

10. Once diagnosis is established, only two procedures seem to be advisable: root canal treatment or extraction.

11. As to prevention, the following recommendations are made: Careful cavity preparation is essential. The use of cavity varnish seems to be indicated in every cavity. In deep cavities, in addition to the cavity varnish, an oxyphosphate cement base should be placed under amalgam fillings.

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ETIOLOGIC FACTORS IN DENTOFACIAL ANOMALIES

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INTRODUCTION

AN understanding of the etiology of dentofacial anomalies is fundamental to orthodontic progress. The more clearly we see the causative factors in malocclusion, the more efficient we can be in the exercise of preventive measures, thereby reducing the necessity of mechanical treatment. One prerequisite to the study of the etiology of dentofacial deformities is an understanding of the normal developmental mechanics; a second prerequisite is the appraisal of environmental influences.

THE LITERATURE

Investigation of growth and development has been active and prolific. Contributors to this field include Atkinson,[1, 2] Brasch,[3] Broadbent,[4, 5] Brodie,[6, 8, 9] Engel,[7] Diamond,[10] Hellman,[11, 12, 13] Herzberg,[14] Higley,[15, 16] Hrdlicka,[17] Keith and Campion,[18] Korkhaus,[19] Krogman,[20] Margolis,[21, 22, 23] McDowell,[24] Todd[25, 26] and Thompson.[27] In spite of the extent of painstaking investigation, a complete picture of developmental mechanics is not available at present because of the amount of time and material necessary to this type of investigation.

Contributors to the specific field of the etiology of malocclusion include Baker,[28] Brown,[29] de Coster,[30] Dewel,[31] Futterman,[32] Johnson,[33, 34] Moore,[35] Salzmann,[36] Sly,[37] Stillman[38] and Waugh.[39] Thus we find ourselves confronted with a mass of valuable material that must be organized and correlated to be of maximum usefulness.

METHODS AND MATERIALS

There are three possible approaches to clarification of the normal developmental mechanics of the masticatory apparatus. One widely employed approach is the measurement and study of normal material and, by comparison of a multitude of cases over a period of years, an endeavor to establish the norm. This has been practiced extensively during the last decade.

A second approach to clarification of the biomechanics is a study of pathology. Frequently, the true nature of the norm is determined through the recognition and understanding of abnormality and its origin. A third possible approach is animal experimentation. This, however, is in reality only the application of the second approach; i.e., a study of abnormal conditions artificially created in animals.

This paper is based partly upon experimental, but mostly upon clinical, observation of the abnormal and particularly of the factors in abnormal structure of the human jaws and abnormal tooth alinement.

FINDINGS

The experimental evidence upon which this paper is based has been published.[40, 41, 42] The experimental results demonstrated the definite influence of environmental conditions on the position and shape of the jaws and teeth. The experiments on *Macacus rhesus* monkeys

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included the application of orthodontic appliances, changes in the vertical dimension, extraction of teeth and artificially created pathologic conditions in the masticatory muscles. The resulting bone changes in the jaws and temporomandibular joint could be demonstrated histologically and, together with clinical observations, form the basis for a general discussion of the environmental factor in the etiology and treatment of malocclusion.

The experimental findings are complemented by the following clinical observation: A number of cases of malocclusion have shown spontaneous improvement as soon as the mechanical fixation, which, as we know now, prevented improvement, has been eliminated. The models and history of some of these cases are presented in Figures 6 to 9. Since this paper was written, the number has increased considerably.

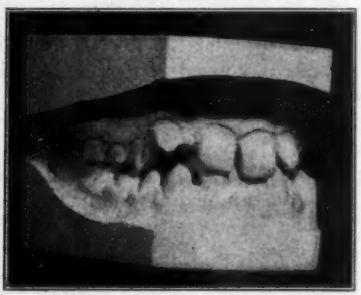

Fig. 1.—Case in boy aged 12 years. The origin of the malocclusion can be traced to eruption of both upper lateral incisors lingually from the lower central incisors. The lower right lateral incisor was then caught lingually from the upper right lateral incisor. Forward migration of both upper lateral segments and the right lower lateral segment followed. Release of the original mechanical impediment would very likely have prevented malocclusion, which, at this point, requires extensive treatment by appliances. (Compare Figure 3.)

DISCUSSION

Among the factors which determine the final shape of the masticatory apparatus, an inherited growth pattern plays the dominant rôle. During intrauterine life, the inherited factors are almost undisturbed; but, from birth on, a multitude of functional and environmental influences are superimposed on the inherited pattern of development.

According to these facts, we may look for the etiology of deviations from the norm mainly in (1) damaging functional and environmental influences and (2) disturbances of developmental mechanics. (Table 1.)

TABLE I.—CLASSIFICATION OF ETIOLOGIC FACTORS IN MALOCCLUSION

1. Damaging functional and environmental influences.
   Disturbances of functional equilibrium.
2. Disturbances of developmental mechanics.
   A. Permanent.
      1. Systemic.
         (a) Congenital; e.g., endocrine disturbances, syphilis.
         (b) Postnatally acquired; e.g., arthritis.
      2. Local.
         (a) Congenital; e.g., aplasia, supernumerary teeth.
         (b) Postnatally acquired; e.g., extraction, mutilation, caries.
   B. Temporary.
      1. Systemic; e.g., acute infection, malnutrition.
      2. Local; e.g., disturbed spatial or temporal relation between jaw and tooth development.

1. Damaging functional and environmental influences on normal development and their recognition and elimination have so far constituted the main endeavor of orthodontic prophylaxis. The recognition of damaging environmental conditions is important because it enables us to attempt elimination of these factors in our efforts to prevent malocclusion. Some of the functional and environmental influences superimposed upon the inherited growth pattern are understood; but the possibilities of en-

vironmental and functional influences are practically innumerable. There are all the factors in normal function, such as feeding and mastication, breathing and speech, as well as other functional habits which we are not accustomed to call "habits" because they are functional. There are, in addition, many possibilities of acquired bad habits which we are accustomed to summarize under the caption "habits." Furthermore, all the tissues surrounding the jaws, such as the connective tissue and muscles attached or adjacent to the jaws, exert an influence upon the hard tissues. Then there are additional factors to be counted among the environmental ones, resulting from a host of possible pathologic changes in the environment of the jaws. It seems almost futile to attempt to classify the functional and environmental influences on the final product; that is, the shape of the adult jaws.

The attempts to describe some of the environmental factors and to study their effects on the dentition are numerous. The most obvious environmental influence, that is habits leading to malformation, has attracted the most attention. A comprehensive article on the subject was published by A. L. Johnson.[34] The importance of normal function has been emphasized throughout orthodontic literature. By studying individual environmental factors, general conclusions can hardly be drawn. An analysis of the multitude of possible environmental factors can, however, be furthered by the application of a fundamental theory governing all environmental influences throughout the body:

Every tissue and every organ in the body is part of the organism. Its welfare is biologically and physically dependent on other organs and tissues within the organism. Every organ is surrounded by other organs which exert a physical influence upon it. It is characteristic of the entire body that a state of equilibrium exists within and between organs. The adaptability to environmental changes of the human body as a whole results largely from the capacity of organs to change their shape rapidly as soon as this equilibrium is disturbed in any way. These changes take place with varying speed in various organs, until the equilibrium is reestablished. (Bone seems to be the most adaptable of the tissues.) This state of equilibrium is essential to the maintenance of the structure of any part of the body. We may term it functional equilibrium.[42, 43]

As was said before, this theory holds true for any part of the body and we may apply it to the masticatory apparatus in the following manner:

A variety of forces are constantly act-

Fig. 2.—Occlusal view of cast shown in Figure 1.

ing upon the masticatory skeleton. It is these forces which constitute what we call environmental influences. Many of these forces are not under the control of the individual will, and the majority of them are responsible for function and dysfunction. Almost all these forces act upon the hard structures from the outside and, therefore, are plainly environmental. A part of the environmental forces act directly on the bone, being attached to it, as are the masticatory muscles. Another part of these forces form a sort of envelope or wrapper around the skeleton, as do the facial muscles, and still another part of these forces acts upon the lingual side of the jaws, as does the tongue or the vacuum in the closed mouth. Some of these forces antagonizing each other and acting upon the masticatory skeleton are listed in Table 2.

To maintain its shape, all of the forces acting upon the skeleton must counteract one another in perfect balance. Any disturbance of this balance will cause bone transformations, which will continue as long as the balance is not reestablished. Growth can be considered from this point of view as one of the active forces. In a growing individual, the growth impulses are added to the otherwise balanced arrangement, creating imbalance and change of shape as long as they exist.

With this theory in mind, we shall be successful in explaining many physiologic or pathologic occurrences. A few examples may serve as illustrations. First, the simplest example:

An orthodontic appliance with an acting spring represents a force added to the previous arrangement and one that will cause bone changes leading to a reestablishment of balance. Commonly speaking, a tooth affected by a spring will migrate as long as the spring is active; but the spring remains only one of many forces acting upon that tooth.

By the same token, a tooth that has been moved in this manner will have the tendency to return to its original position after the removal of the spring. Here is the answer to the orthodontist's retention problem. Unless natural forces take over the action of the spring or of a mechanical retainer, the result of a tooth movement will never be stable. This

TABLE 2.—SOME ANTAGONISTIC FORCES ACTING UPON MASTICATORY APPARATUS.

Lips ——→	←—— Tongue
Cheeks ——→	←—— Tongue
Eruption (growth of teeth) ——→	←—— Masticatory muscles (masseter, temporalis, internal pterygoid)
Air pressure on skin and nasal cavity ——→	←—— Tongue in closed mouth, air pressure in open mouth
Masseter ——→	←—— Elasticity of periodontal membrane, particularly of molars, and suprahyoid muscles
Internal pterygoid	
(a) in vertical movement ——→	←—— Same as masseter
(b) in lateral movement ——→	←—— Internal pterygoid of other side
External pterygoid	
(a) in anterior movement ——→	←—— Posterior third of temporalis, suprahyoid group, digastricus, muscles of the neck
(b) in lateral movement ——→	←—— External pterygoid of other side

theory can, therefore, aid in determining where, when and for how long a time retention is needed.

Another illustration of disturbed functional equilibrium is the bone transformation seen in the jaws and the temporomandibular joint after artificial change of the vertical dimension in experimental animals.[40, 41, 42] Any such change of the vertical dimension alters the mechanical arrangement of the musculature acting upon the jaws. Raising the bite, for instance, will increase the tension of the mouth-closing muscles and relax the mouth-opening muscles. The histologic changes described in previous papers as subsequent to artificial changes in the vertical dimension monkeys, of the validity of the theory.[41] For instance, paralysis of the third division of the fifth nerve caused repositioning of the mandible to a more anterior position. Whatever the cause of this change may have been, its origin can be sought only in the effected muscular paralysis which disturbed the functional equilibrium.

Innumerable examples could be given of how the coordination of a problem with this theory helps in analyzing, understanding and solving it.

2. Disturbances of developmental mechanics comprise a second group of etiologic factors in malocclusion. Such disturbances may be permanent or temporary. (Table 1.)

Fig. 3.—Case in girl aged 10 years. Left: The right upper lateral incisor is in the process of erupting lingually from the lower right lateral incisor. Right: Opening of the bite permitted self-correction within ten days.

can be explained in this manner. The action of biteplates can be determined in correlation with this theory. This has been discussed in previous papers.[40, 41]

Many clinical observations can easily be correlated with this theory. For instance, all bad habits, such as thumb-sucking and lip-biting, represent forces added to the otherwise balanced arrangement. Pathologic changes in the striated musculature, such as are encountered in pseudohypertrophic muscular dystrophy, result in characteristic bone malformation.[32]

There is also striking evidence, in previously published experiments on

Permanent disturbances may be either systemic or local. Permanent local disturbance of developmental mechanics may be either congenital or postnatally acquired. Cretinism or syphilis, for instance, represents a systemic disturbance. Supernumerary, malformed or missing teeth represent a local disturbance of the permanent group.

A permanent disturbance of developmental mechanics may be not congenital, but postnatally acquired. Arthritis, for instance, affecting the normal development and function of the mandible, represents a systemic disturbance which is acquired and is often followed by perma-

nent damage. Extraction of a permanent first molar during development represents a local acquired permanent disturbance of developmental mechanics. Loss of tooth material through caries or trauma belongs in this group.

Most permanent disturbances of developmental mechanics, congenital as well as acquired, are easily recognized, for systemic factors usually manifest themselves in symptoms additional to those found in the masticatory apparatus, and local factors are easily diagnosed. Congenital systemic factors are, however, difficult to cope with. Too little is known about them to afford practical treatment. Important investigations in this field have been conducted by Schour and Massler[44] and Ziskin.[45]

developed, represents a local discrepancy of timing.

(In the classification given in Table 1, the term "hereditary" was purposely avoided. While it cannot be denied that certain anomalies may occur in consecutive generations of one family, the visible anomaly in itself may not be hereditary at all, but may rather originate in many possible causative conditions. The anomaly that manifests itself is, in most cases merely the last link in what may be a long chain of interdependent factors, one or all of which may be hereditary. For instance, a protrusion of the upper incisors "running in a family" can be traced to underdevelopment of the mandible, but this, again, is not necessarily hereditary. The condition may be hered-

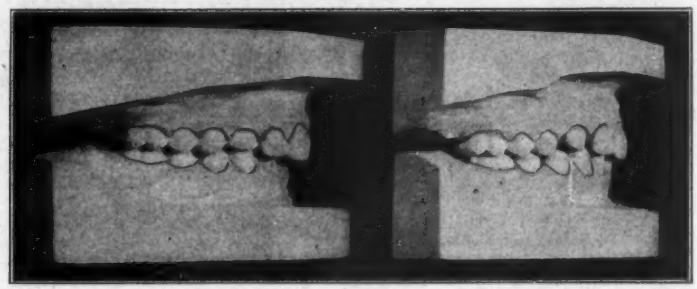

Fig. 4.—Developing Class II malocclusion in girl aged 11 years; right view. Widening of the upper arch and raising of the bite (accomplished by the use of one split biteplate) permitted improvement of the relation of the upper and lower dentition. Casts at the right in Figures 4, 5 and 6 were taken four and one-half months after those at the left.

A temporary disturbance of developmental mechanics may be systemic or local. Temporary malnutrition or acute infection is systemic. Most temporary disturbances are, however, postnatally acquired. These may be systemic or local. A nutritional disturbance like rickets represents an acquired systemic temporary disturbance. A local temporary disturbance represents a discrepancy of spacing or timing. The premature loss of a deciduous tooth represents a local temporary disturbance of space. Delayed bone growth, when the teeth are already

itary or it may have its origin in a dysfunction of an endocrine gland, which, in turn, is dependent on an abnormality of another endocrine gland, and which, in its turn, may be influenced by faulty metabolism caused by other dysfunction; and any one of these may form a hereditary factor. Which of the links is inherited in the individual case it is impossible to ascertain. Moreover, the manifestation of similar anomalies in consecutive generations may stem from different and unrelated causative conditions. Therefore, the use of the term

"hereditary" as a means of classifying etiologic factors is not particularly useful at our present stage of knowledge.)

The majority of temporary disturbances of developmental mechanics are caused by local conditions. While they are temporary, they often lead through mechanical fixation to lasting malocclusion, and so their transitory nature has remained unrecognized.

It is, however, these local temporary disturbances that should probably take most of the blame for the high incidence of dentofacial anomalies in man. It is interesting to note that mankind is afflicted by a tremendous number of dentofacial anomalies, while the incidence of malformation and monstrosities of the rest of the body is comparatively low. The reason for this fact may be found in a peculiarity of the masticatory apparatus, in that it contains more than one hard structure. Most organs represent a composite of various tissues, often originating from different germ layers. These tissues grow and develop more or less independently of one another, but in harmony with one another, to form the resultant normal organ. We may safely assume that the tissues within one organ do not always grow at the same time to the necessary degree, but that they have growth spurts at various times and of various intensity. Temporary discrepancies may very well occur without interfering with the normal final result because they can and do straighten out spontaneously through the adaptability of the various tissues to one another and through compensating growth during the long period of development. No organ within the body but the masticatory apparatus contains more than one hard tissue; namely, bone. Therefore, mechanical interference of the various com-

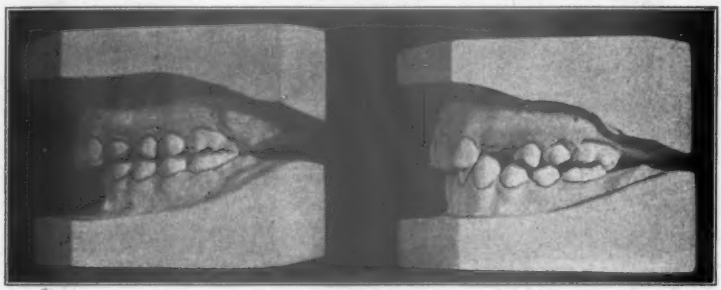

Fig. 5.—Left view of casts shown in Figures 4 and 6.

Fig. 6.—Front view of casts shown in Figures 4 and 5.

ponents threatens only in the masticatory apparatus.

In the masticatory apparatus, we have, aside from bone, connective tissue, muscles and epithelium, and a very important additional hard component, the teeth. For a long period of time, it has not been recognized that the development of the teeth and the development of the jaws are not interdependent. It has been emphasized, with few exceptions (de Coster[30]), throughout the literature that the growth of the jaws is dependent on the development of the teeth, and vice versa. This interdependence has been overestimated and a misconception has been perpetuated throughout dental literature, while any one could observe, on the one hand, perfectly normal development of all individual teeth within jaws having inadequate space and, on the other hand, growth of jaws to normal or almost normal size where one or a number of tooth germs were lacking (anodontia cases with normal faces). (Brodie and Sarnat,[46] Brodie [47] and Feinman.[48]) How important it is to realize the independence of tooth and jaw development has only recently been thoroughly demonstrated by the contributions of M. Diamond.[10]

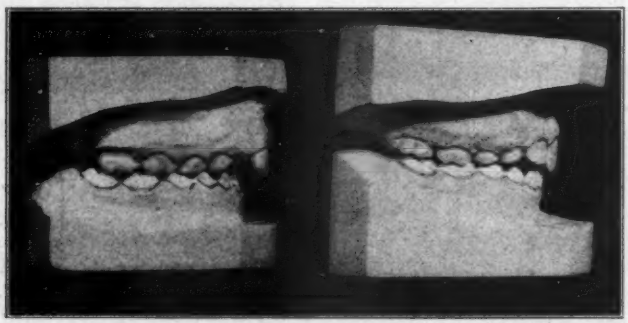

Fig. 7.—Case in girl aged 8 years at time first casts were made (left); Class II malocclusion. Self-correction of the jaw relation within two years was due to elimination of mechanical interference. A split biteplate was used to widen the upper arch and to eliminate overbite.

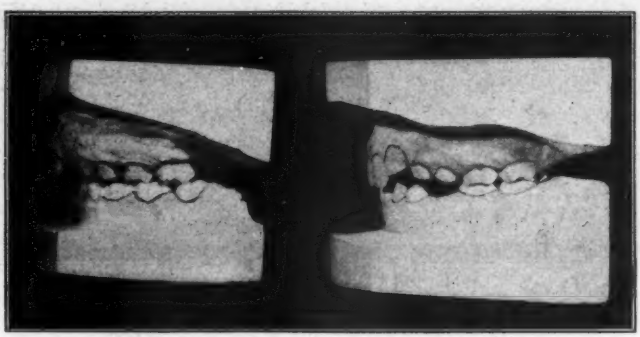

Fig. 8.—Left view of casts shown in Figures 7 and 9.

It is not surprising that where two hard structures (bone and teeth) develop in such close physical relation, but independently of each other, temporary discrepancies are apt to occur.

These discrepancies, originally tempo-

rary in nature, are often made permanent through mechanical fixation. A few examples are as follows:

If the maxilla does not widen in time, a lateral incisor may erupt at the place of its original formation in the jaw; i.e., lingually. The tooth would undoubtedly move into its proper position as soon as the maxillary width increased adequately; but, before this can happen, the maxillary lateral incisor may be caught lingually from the lower incisors, which may have erupted previously, and the lingual occlusion of the upper lateral incisor remains permanently fixed. (Fig. 1.) A similar phenomenon can occur in the mandible if the overbiting upper incisors represent a mechanical obstacle to the straightening out of crowded lower incisors. An analogous condition may occur in the buccolingual relation of the upper and lower first permanent molars.

Such disturbances, caused by lack of circumferential growth of the jaws, lead to Class I Angle malocclusion.

The degree of overjet and overbite, which in itself may be responsible for subsequent development of malocclusion, is partly dependent on the timing of growth spurts in the mandibular ramus. (Diamond.[10]) Other factors also play a rôle in the determination of the incisal overbite.

If, however, an excessive overbite exists at the time of the eruption of the upper and lower first permanent molars, it may very well lead to abnormal interdigitation of these teeth, and subsequently to the development of a Class II malocclusion which represents a permanent condition, unless interfered with.

These and other examples show how a local disturbance of developmental mechanics, originally temporary in nature, may become permanent through mechanical fixation and points the way toward a more active practice of orthodontic prophylaxis.

There are theoretically two ways of influencing growth: one, growth promoting; the other, growth permitting. While our present knowledge does not enable us to promote growth in orthodontic practice, we already have the possibility of permitting or making possible growth that, we know, should occur, but which does not occur because of mechanical impediments. It is the removal of such impediments which we may call a growth-permitting influence and which should become one of the foremost objectives in efforts at prevention of malocclusion.

Once a temporary malrelation of teeth becomes mechanically fixed, we speak of "mechanical interference," which has long been recognized as a damaging condition. (Higley,[16] Rogers[49] and Breit-

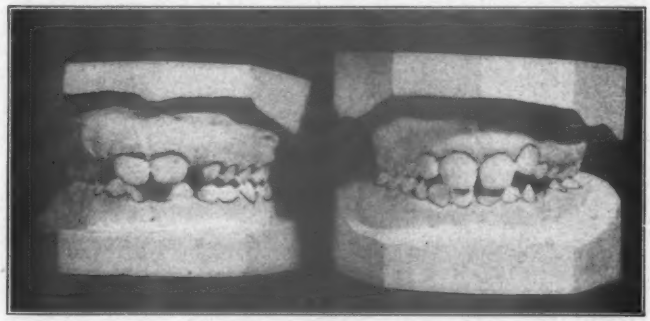

Fig. 9.—Front view of casts shown in Figures 7 and 8.

ner.[50]) It is known that it prevents the spontaneous proper repositioning of individual teeth, and it is also known that it may be the cause of many cases of Class III malocclusion, which, for this reason, have even been differentiated from "real" (systemic) Class III cases. Incidentally, this differentiation is equally applicable to Class II malocclusion. It is known that the removal of mechanical interference must be the first step in mechanical orthodontic treatment and also of myofunctional treatment as advocated by Rogers.[49]

What has not been generally recognized is that mechanical interference, by establishing a local disturbance of developmental mechanics, may in itself be responsible for a great number of cases of malocclusion including Class II Angle cases. Elimination of mechanical interference, however, will often permit self-correction of the anomaly, other treatment, mechanical or myofunctional, proving superfluous.

It is not within the scope of this paper to describe in detail the various temporary local discrepancies of timing and spacing which may occur during development and which, through mechanical fixation, may lead to permanent malocclusion. The following observations should, however, be mentioned here:

Many Class I Angle cases are certainly due to a temporary disturbance of local character. Some Class III Angle cases probably have their origin in a temporary disturbance of the incisal overbite relation that was not corrected in time. Other Class III cases are of a definitely systemic character. The origin of a great majority of Class II Angle cases can be traced to a temporary growth disturbance during the establishment of the incisal overbite and eruption of the first permanent molars. Few Class II cases exist in the deciduous dentition, and their incidence suddenly rises with the eruption of the permanent teeth. That the disturbance had been only temporary is evidenced by possible self-correction after release from mechanical interference.

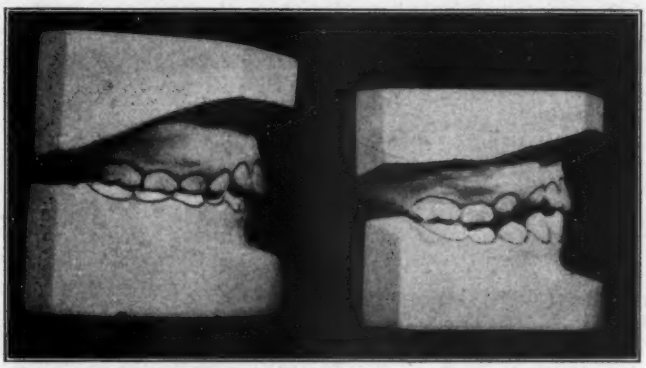

Fig. 10.—Case in girl aged 10 years at time first casts were made (left); Class II malocclusion. Self-correction was accomplished within one and one-half years through elimination of mechanical interference as in Figures 1-9.

Figures 2 to 5 represent cases that, by simple release of the existing mechanical fixation, improved spontaneously. All of these cases, lacking intervention, would undoubtedly have persisted and deteriorated.

We may, therefore, safely assume that a growth tendency toward the norm

existed in these cases, and that, given the opportunity, self-correction occurred.

The fact that such self-correction may occur must be considered significant in our endeavor to reduce the incidence of dentofacial anomalies to be found at the age of completed dentition.

It is evident that such prophylactic means as are advocated in this paper should be instituted as soon as the growth-impeding condition is recognized; that is, long before the completion of the permanent dentition. The practice, advocated by some orthodontists, of postponing any orthodontic interference until after 11 or 12 years of age should be generally replaced by close observation of the developing dentition when growth of jaws liberated from mechanical interference can still be expected.

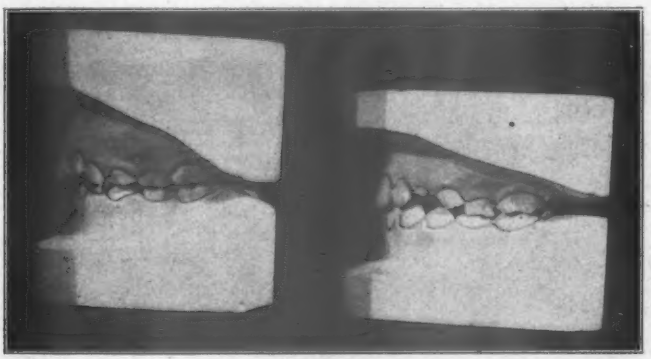

Fig. 11.—Left view of casts shown in Figures 10 and 12.

SUMMARY

An understanding of the etiology of dentofacial anomalies is fundamental to orthodontic progress. The causes of dentofacial anomalies are chiefly twofold: (1) damaging environmental influences and (2) disturbances of developmental mechanics. Orthodontic prophylaxis has so far concentrated upon the elimination of some damaging environmental influences; for our present knowledge is insufficient to attempt modification of the inherited pattern of development in practice. Most disturbances of developmental mechanics, however, seem to be temporary rather than permanent. There exist two developmental processes, occurring simultaneously, but independent of each other, which must be perfectly correlated in time and space in order to create a normal masticatory apparatus. In such a complicated arrangement, temporary disturbances may frequently occur. While in other organs such temporary disturbances may cease spontaneously, it is characteristic of the dentition that a temporary disturbance usually becomes permanent through mechanical fixation. This explains the great number of cases of malocclusion in mankind, and it also points to successful self-correction of such cases through elimination of any mechanical interference that hinders normal development. There is clinical evidence that malocclusion which exists between the ages of 8 and 10 and which certainly would persist throughout life unless interfered with, disappears spontaneously if unimpeded growth is permitted through elimination of mechanical hindrances. It is evident that such measures as are advocated in this report are to be instituted during the period of accelerated growth; that is, between the ages of 5 and 11 years.

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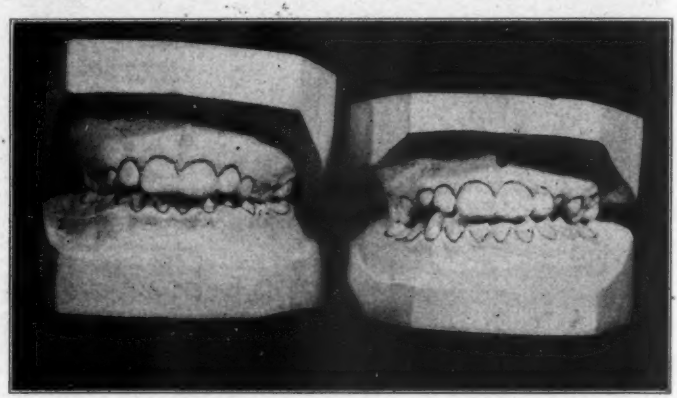

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RECOMMENDATIONS FOR EXPERIMENTAL PREPAYMENT DENTAL PLAN

COUNCIL ON DENTAL HEALTH
AMERICAN DENTAL ASSOCIATION

THE past decade has witnessed many changes in the economic and social lives of the American people. Among other things, these changes have affected the fields of health, hospital, medical and dental economics. Voluntary nonprofit prepayment hospital plans have had a phenomenal growth during this period, and recently we have seen a similar development of plans for prepayment medical care. The growing demand for a wider distribution of health services has had its reaction in many interested groups and agencies, both public and private, as well as in federal and state legislative bodies.

Dentistry is assuming an important role in contributing to the health of the public. The dental profession is concerned with the development and elevation of its standards of service. The profession is also seriously interested in making this service available to a greater number of people.

Through the efforts of organized dentistry, dental health service in this country has reached a level unsurpassed in quality and quantity by that of any other nation. The distribution of dental service to those able to pay prevailing private practice fees without financial hardship need not present any problem. Satisfactory dental care programs for the indigent group have been developed and are being expanded. However, there remains a large segment of the population in the medium and low income groups for which a more satisfactory dental service should be provided.

The Council on Dental Health of the American Dental Association recognizes that a lack of understanding of the relationship between dental and general health, the fear of pain and procrastination have caused a certain percentage of the population to neglect their dental needs.

As a result of its interest in this problem and in line with the general policy of broadening the scope of health services to the public, the American Dental Association created the Council on Dental Health, and charged it, among other responsibilities, "to study the need and develop plans and programs for the provision of more adequate dental care for the public." One of the important phases of its work has been to study methods of meeting dental care costs and the possibility of applying the prepayment principle to dental service. Studies conducted along these lines have convinced the Council on Dental Health that the American people need not only better distribution of dental service, but also a method whereby the cost of such service may be paid for on a budget or prepayment basis.

The success of budgeting hospital and medical care costs during the past decade is evidenced by the increased demands of the public for further prepayment planning in the health field. To date, more than 15,000,000 persons have enrolled in nonprofit hospital service plans and more than 1,500,000 in nonprofit medical prepayment plans operated under the sponsorship of state and

local medical societies. The records show a steady increase in their growth and expansion. This success of prepayment planning for health services and a public awakening to the value of dental health plus the unpredictable factors in dental care costs are responsible for the growing interest in similar provisions in our field. It is thought that a plan for budgeting the cost of dental service should:

1. Provide a dental service that will contribute substantially to a higher level of dental health for its members.

2. Assure people in the medium and low income brackets of dental service without financial hardship.

3. Provide adequate fees to the dental profession for their services and stabilize the income of the profession.

4. Preserve the private practice of dentistry.

The Council on Dental Health is mindful of the fact that in the formation of a prepayment plan for dental care, so far as the insurance factor is concerned, dental care costs and related problems are not comparable in the true sense of the word with those of either hospital or medical care. It is believed that an individual who visits his dentist regularly and receives adequate dental care can budget the cost of his dental needs to a certain extent, whereas medical and hospital care costs are less predictable. Despite the lack of similarity among dental, medical and hospital care problems, the Council on Dental Health was able to gain considerable information from a study of the development of medical and hospital service plans. However, as there is not sufficient factual or statistical material to determine the feasibility of prepayment planning in dentistry at the present time, the only means by which we can know whether a prepayment plan for dental service is possible is actual experiment.

In consideration of these facts, the Council on Dental Health made a study of a majority of the existing medical service prepayment plans and of a number of existing hospital service prepayment plans. As a result of these studies, it is recommended that experimental dental service prepayment plans be inaugurated under the direction or supervision of component dental societies, under the following conditions:

1. The city or political subdivision selected should be of not less than 100,000 population.

2. The trial period should be twelve months.

3. The enrollment should not exceed 5,000 persons during this trial period.

4. The enrolled persons should represent a cross-section of employed groups within certain income limitations.

5. The plan should be offered only to groups of ten or more employed persons and through their place of employment on a payroll deduction basis.

6. A minimum of 60 per cent of the persons in an employed group should be enrolled.

7. Sound enrollment procedures as followed by existing voluntary nonprofit hospital and medical service plans should be adopted.

The successful application of the prepayment principle in dentistry will depend largely on the dental needs of subscribers, on the amount paid by the subscriber, on the types and amounts of service provided under the plan and on the fee schedule of the participating dentists. The main objective of the experimental program is to obtain data on the relationship between these factors in a dental prepayment plan. It is proposed that the experimental program be financed by an underwriting fund in order that the participating dentists may receive the prevailing fee for service rendered in case the amount collected from the subscriber does not meet the actual costs during the experimental period. When actuarial data have been obtained, the monthly payment by the subscriber can be adjusted to make the

prepayment plan self-sustaining. In addition, if surplus monies accrue, additional dental service benefits may be offered to the subscriber, since the plan will be operated on a nonprofit basis.

The recommended legal instruments to be used in connection with a dental prepayment plan consist of articles of incorporation, bylaws, the subscriber's application for a dental service certificate, the subscriber's dental service certificate and an application for registration as a participating dentist.

ARTICLES OF INCORPORATION

The articles of incorporation establish a nonprofit corporation called the (name of state) dental service, to (1) maintain and operate a voluntary nonprofit dental service plan, (2) accept charitable or benevolent trust gifts or legacies or devices intended for the benefit of public health and (3) collect statistics and data in connection with the operation of the plan that may be deemed of value to the community and to the dental profession.

The articles of incorporation provide that the membership of the corporation shall be composed of licensed dentists who are members in good standing of the state dental society and of such other persons as the board of trustees of the corporation shall elect to membership to serve as trustees, provided that at least two-thirds of the membership of the board of trustees shall be composed of dentists. The articles of incorporation further provide that the corporation shall be organized upon a nonstock basis and that it shall be financed from monthly payments received from individuals or groups of individuals who purchase dental service under the plan and, if necessary, from underwriting funds or contributions.

BYLAWS

The bylaws establish provisions for the annual and special meetings of the corporation and the qualifications for membership on the board of trustees.

In addition to supervision and control of the business affairs of the corporation, the board of trustees has the authority to decide the scope of the services to be furnished subscribers, to adopt rates and fee schedules, to incur indebtedness under certain conditions, to invest funds and to delegate its powers to committees and officers of the corporation.

Provisions are established to determine the eligibility of subscribers and of dentists who wish to be registered with the corporation. The corporation does not assume the liability of a dentist arising from the dentist-patient relationship.

A dentist rendering service to a subscriber cannot make a direct charge to the subscriber for such services as the subscriber is entitled to under the plan, but payment is made through the corporation. Additional services, however, are financed through the arrangements made by the patient and the dentist.

SUBSCRIBER'S APPLICATION FOR DENTAL SERVICE CERTIFICATE

The formal request for dental service under the prepayment plan is made on a form provided for that purpose. The application authorizes the employer to deduct the monthly fee from the subscriber's wages. The amount of the monthly fee, which is established by the corporation, varies with the income bracket classification and number of dependents.

SUBSCRIBER'S DENTAL SERVICE CERTIFICATE

If the subscriber is accepted by the corporation, a dental service certificate is issued to him upon the payment of a registration fee of $1 plus the monthly fee. The certificate constitutes a contract between the corporation and the subscriber. The subscriber and his dependents then become eligible for the dental services that are specifically pro-

vided in the plan. Eligible dependents are spouse and unmarried children under 19 years of age living with and dependent on the subscriber for maintenance and support.

Dental services to which subscriber and dependents are entitled during the experimental period include (1) dental examinations, limited to two per calendar year per person, (2) prophylaxis, limited to two per calendar year per person, (3) extractions, including the extraction of impacted teeth, (4) fillings, when amalgam, silicate and other cements are used, (5) treatment of diseases of the mouth, limited to patients under 19 years of age, (6) emergency treatment for the relief of pain, (7) treatment of jaw fractures and (8) x-ray service.

The following services are not included: (1) denture, bridge, inlay, crown, orthodontic and general anesthetic, and house calls; (2) any service covered by workmen's compensation or employer's liability laws; (3) service rendered by or through any federal, state or charitable agency; (4) service covered by reason of recipient's qualifying for veterans' benefits or benefits for dependents of veterans, and (5) dental service for a disease or injury to a subscriber or dependent for which he is confined to his home or to a hospital, and for which he is under treatment or anticipates the need for treatment at the time that application is made for this certificate.

If, in an emergency, while the subscriber or a dependent is outside the area regularly served by the corporation and not within reasonable access to a participating dentist, service covered by the certificate is rendered by a dentist not registered by the corporation, the corporation will pay to the dentist the prevailing fee established by it.

The subscriber or his dependent has the right to select any participating dentist. A participating dentist shall have the right to decline or accept such subscriber or his dependents in accordance with the custom and practice that prevails in the private practice of dentistry.

The corporation reserves the right to change the rate and benefits on thirty days' written notice to the subscriber. The certificate is issued for a period of twelve months and is renewed automatically on a year-to-year basis subject to the right of cancellation by either party on thirty days' written notice.

As has been stated above, the amount to be paid by the subscriber and the types of service included in the plan are established by the board of trustees of the corporation. The board of trustees also determines the eligibility of the subscriber on a basis of income. The income limitations might vary in different localities and would be determined by local conditions. The following income limitations are presented here as examples: Persons eligible for dental service might be (1) a subscriber without dependents and whose net annual income exceeds $1,800; (2) a subscriber who has one dependent and whose net annual income together with the net annual income of his family exceeds $2,400, or (3) a subscriber who has two or more dependents and whose net annual income together with the net annual income of his family exceeds $3,000. The net annual income is determined by an estimate of family income from all sources for the current calendar year. Participating dentists may make a charge to the subscriber for services not included in the certificate.

APPLICATION FOR REGISTRATION AS PARTICIPATING DENTIST

All dentists who are licensed to practice in the state in which the prepayment plan is in operation and who are members in good standing of the state dental society are eligible to participate in the prepayment plan. Participation, which is voluntary, may be terminated by written notice at least ninety days prior to the

effective date, provided, however, that any dental service undertaken for a subscriber shall be completed in accordance with the terms of the subscriber's dental service certificate.

The participating dentists receive payment from the corporation for the dental services included in the plan and in accordance with the fee schedule. The subscriber may elect to receive additional service (example, inlay or denture), in which case a separate financial arrangement is made between the patient and the dentist.

In presenting this prepayment plan as an experimental program, cognizance must be taken of the fact that local laws and economic conditions would prevent the adoption of a uniform program throughout the country. The local dental society can best determine what provisions should be made at the outset or what adjustments may later be indicated in the subscriber's monthly payment, in the amount of service to be provided under the plan, in the subscriber's income limitations and in the age limit for dependents.

In establishing the subscriber's payment rate, adequate provisions should be made for overhead expense and a reasonable contingency fund. The rate should also be consistent with present payments by the public for dental service in the area.

It is also important to emphasize that the state or local dental society will have the responsibility of establishing a fee schedule for the dental service to which the subscriber is entitled under the plan. These matters, of course, are details which can be given consideration as the plans assume a more concrete form.

SUMMARY

The need for finding a more satisfactory financing plan to meet dental care costs, especially for the medium and low income groups, suggests the desirability of establishing experimental prepayment dental plans. Although the successful application of the prepayment principle in meeting hospital and medical care costs has been well established, present knowledge of the subject is inadequate to determine whether a similar budgeting plan can be applied to dentistry.

It is recommended that experimental dental service prepayment plans be inaugurated by dental societies. With proper management, it is believed that actuarial data can be obtained to analyze the now unknown factors of a prepayment system as they are related to dental care.

Copies of the proposed experimental prepayment dental plan, which contain sample legal instruments, can be secured upon request from the Council on Dental Health of the American Dental Association, 222 East Superior St., Chicago 11, Ill.

SOLITARY NEUROFIBROMA OF THE ORAL CAVITY (NEURILEMMOMA; NEURINOMA)

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SOLITARY benign tumors arising from the peripheral nervous system have been observed frequently in different parts of the body. They occur most frequently along the posterior divisions of the spinal and acoustic nerves and less commonly along the other cranial nerves. Stout,[1, 2] in reviewing the subject, says that, from 1926 to January 1944, there have been recorded in the laboratory of surgical pathology of Columbia University 130 cases of neurilemmoma. Only two of these cases involved the oral cavity and pharynx.

Recently Smith,[3] reporting on three cases observed in the larynx, mentioned that only twelve other cases are recorded in medical literature to date; while Bogdasarian and Stout[4] report the only observed case of neurilemmoma of the nasal septum. In dental literature, Zilken[5] reported a case occurring in the lower jaw and, in his review of European literature, could find but few oral cases.

I wish to report two cases observed by me and also record the case treated by F. M. McCaffery, which I observed at the time. This case was included in Stout's series.

REPORT OF CASES

CASE 1.—F. P., a white youth, aged 19, presented a swelling in the roof of his mouth and complained of pain. About seven years before, he first noticed a very small lump in about the center of the palate. There were no complaints until about three months earlier, when he experienced some pain. The lump had also increased in size. During the years, several biopsies had been made, with a diagnosis of fibroma or fibrosarcoma.

The specimen removed by Dr. McCaffery was reported on by Dr. Stout, October 25.

Section showed that the tumor was made up of an admixture of about equal parts of Antoni type A and type B tissues. The type A tissue was well differentiated, with well-developed palisading of nuclei and twisted Verocay bodies. The type B tissue showed microcystic degeneration and was obscured in places by severe inflammatory cell infiltration, which partly obscured its architecture and which was probably the result of superficial ulceration. At its periphery, the tumor was encapsulated. This was a characteristic example of a neurilemmoma arising, no doubt, from one of the small nerve twigs of the region.

CASE 2.—*History.*—A woman, S. C., aged 29, admitted to Mt. Sinai Hospital, service of Dr. Ralph Colp, complained of a tense swelling in the left submaxillary region of one year's duration. The swelling had varied in size. There was no relationship to eating. Several months earlier, the patient went to her dentist and he extracted the lower first molar tooth, but no improvement of the condition resulted. The general history was negative.

Examination.—Examination of the mouth and jaw revealed a walnut-sized swelling palpable through the floor of the mouth. No calculus concretions were felt or seen roentgenographically. No evacuation from the duct was observed when the gland was milked. The mouth was dry. The general physical examination was negative.

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Diagnosis.—The preoperative diagnosis was submaxillary cyst.

Operation.—August 27, 1936, under local conduction anesthesia, a 3-inch longitudinal incision was made through the mucous membrane of the floor of the mouth to the left of the tongue. The mucous membrane was separated from the underlying mass and the mass from the underlying gland. The wound was then sutured. The mass removed was 1½ by ¾ inches in size, kidney-shaped, distended and semisolid. On section, it had a whitish solid glistening appearance.

Pathologic Report.—(Dr. Klemperer.) The slide showed five different slices of a well-encapsulated tumor. In all parts, the histologic appearance was the same. The tumor was made up of large areas of cells

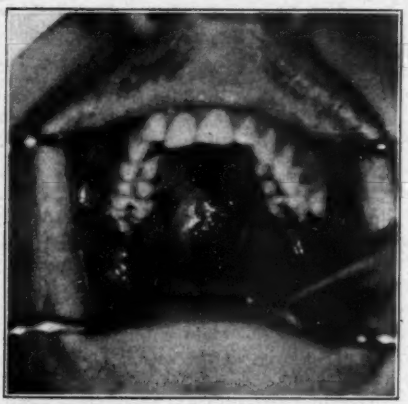

Fig. 1 (Case 1).—Case observed with Dr. McCaffery and included in Dr. Stout's series. (October 1934.)

of elongated shape, showing well-defined alinement of nuclei to produce the characteristic palisade effect. These large masses of Antoni type A tissue are often arranged in a twisted nodular manner so as to produce the appearance of so-called Verocay bodies. Between the large masses of type A tissue were much smaller insignificant areas of type B tissue with occasional microcystic degeneration. The blood vessels were usually found in the type B areas. Very few of them had thick collagen sheaths. Nowhere did the tumor invade its capsule.

This was a characteristic neurilemmoma composed very largely of solid type A tissue and with so little evidence of microcystic degeneration that one would not expect to be able to note its presence on gross examination.

Diagnosis.—The diagnosis was neurinoma of the floor of the mouth.

Course.—The course was uneventful.

CASE 3.—*History.*—Mrs. B. B., aged 48, was referred to the oral surgery department of Columbia University because of a swelling in the buccal parieties of the left cheek, of one month's duration. The swelling unseated the patient's dentures.

Examination.—The patient was edentulous. In the posterior region of the left buccal parieties was a moderate-sized soft swelling, between the upper and the lower

Fig. 2 (Case 2). — Neurinoma removed August 27, 1936.

jaws and projecting into the oral cavity. Palpation of the mass produced a painful sensation at the mental region of the jaw and the lower lip. The overlying mucous membrane was normal in color and not inflamed.

Roentgenographic examination of the left side of the mandible revealed an area of decalcification involving the entire ramus of the mandible.

Preoperative Diagnosis.—The diagnosis was cyst of the mandible.

Operation.—November 13, 1943, under general anesthesia, an incision was made along the anterior surface of the ramus of

the mandible and the cyst membrane was enucleated from the jaw bone. Soft bright orange crystalline matter was present in the fluid of the cyst. The wound was sutured and an iodiform gauze drain inserted.

Pathologic Report.—Gross: The specimen consisted of a piece of tissue 3 by 2.5 by 2 cm. and many small pieces of bright orange material which was very soft. The main piece of tissue was encapsulated, dark red and soft. Within the capsule was more of the soft orange material.

Microscopic: Section showed that there was a dense capsule of fibrous tissue showing many large lymph vessels and occasional clumps of inflammatory cells, mainly lymphocytes and polymorphonuclear leukocytes. This passed over more or less gradually into fibrous-looking tissue, the nuclei of which were larger and more closely approximated to each other. Very few mitoses were seen. Between these areas of elongated cells were clumps of large pale foam cells, some of which also contained pale green highly refractile granules, which might have been hemosiderin. The tumor cells tended to be arranged in strands, which ended in whorls. Palisading of the nuclei was seen in several places. There were some areas where necrosis of the tissue seemed to have occurred. (This is no sign of invasiveness.)

Diagnosis.—Neurilemmoma of the ramus of the left mandible was diagnosed.

(This unexpected and unusual finding, I suppose, must have had origin in the sheath of the inferior dental nerve. Degeneration had been extensive, having the effect of the presence of a cyst.)

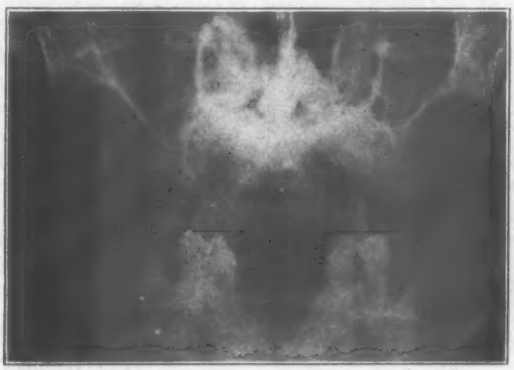

Fig. 3 (Case 3).—Neurilemmoma of mandibular nerve; showing decalcification of ramus of mandible.

COMMENT

Since peripheral tumors of the nervous system are relatively infrequent in dental practice, a review of the subject might not be amiss.

Peripheral tumors of the nervous system have been the subject of intense study by numerous investigators in the past. Much research has been directed to the study of their complex structure and to their origin in supporting cells which accompany nerve fibers, such as the cells of the sheath of Schwann, the neurilemma, the endoneurium and the lymph sheaths of the nerve trunk, all of which participate in tumor growth.

One result of the varying emphasis placed upon different phases of this complex problem is the multiplicity of names given to these tumors by the various investigators: solitary neurofibroma, neurinoma, fibroma of nerves, neurilemmoma, perineural fibroblastoma, gliofibrosarcoma, peripheral glioma, Schwannoma, cerebello-pentile-angle tumor, tumor of nervus acusticus, glioma of acoustic nerve, etc.

There is also a difference of opinion

as to whether these tumors are formed of connective tissue, of nerve tissue or the sheath of Schwann cells.

Virchow believed that these tumors arose from nerve tissue, but because he could not find true nerve cells within the mass called them false neuromas as distinguished from true neuromas. Thomson later observed that there were single and multiple varieties of this tumor, which von Recklinghausen believed had a common connective tissue origin. Verocay, in 1910, offered evidence that the tumor was made up of nerve cells or their embryonal equivalents and suggested the name "neurinoma."

Later, Verocay's followers advanced the theory that the cells of the sheath

Fig. 4 (Case 3).—Lateral view.

of Schwann were the originating factors and suggested the name of "schwannoma."

More recently, Penfield,[6] among others, offered evidence that the tumor arose from the neural connective tissue and that the type cell was a connective tissue cell; this giving rise to such terms as "peripheral fibroblastoma," "perineural fibroblastoma," "peripheral glioma" and "neurilemmoma."

It is commonly accepted now that solitary neurofibroma is usually a single encapsulated tumor with a thin shining capsule. It is usually round, but often presents knobs and is either solid or cystic. It is benign, but may recur locally unless completely removed.

The solitary neurofibroma must be differentiated from the multiple fibromatosis of von Recklinghausen's disease, which is a systemic disease.

Von Recklinghausen's disease is hereditary, manifesting itself by the appearance of multiple encapsulated tumors upon the peripheral nerve or root ends. Other manifestations may be pigmented moles, *cafe au lait* spots and diffuse thickening of the nerve. Multiple fibromatosis of von Recklinghausen is not a true neoplastic process.

SUMMARY

Of two rare cases of solitary neurofibroma observed in oral surgery practice, the first was that of a solid tumor found beneath the mucous membrane of the mouth and situated above the submaxillary gland. In the second case, the lesion, which was cystic, was within the ramus of the mandible. Roentgenographically, it resembled the ordinary large cyst or adamantinoma in the mandible.

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EFFECTS OF INDUCTOTHERMY ON PERIAPICAL FOCAL INFECTION AS EVIDENCED BY CHANGES IN SEDIMENTATION RATE*

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THE fact that the teeth are an integral part of the body and not isolated entities has made an understanding of tissue reactions to root-canal operations very important. Both the dental and the medical profession are interested in the problem of the pulpless tooth as a focus of infection. Much has been written during the last few years on this problem, yet there is need for more satisfactory methods of studying the relationship of the pulpless tooth to general health.

There have been many approaches to the study of the pulpless tooth problem. One of the most important methods of study requires the extraction of the tooth in question to determine the effect of its removal on the condition of the patient and to make histologic studies of the periapical tissues possible. This method is not satisfactory since it requires the removal of the tooth before it is proved guilty. A valuable organ of mastication is lost needlessly if histologic examination of the tooth shows signs of repair or normal tissue. The diagnosis of focal infection today is instead based on roentgenographic interpretation alone. Outstanding contributors who have evolved methods of studying this problem include Rhoads and Dick, Haden, Rosenow, Eusterman, Blayney, Coolidge, Hatton and Kronfeld.

In the studies conducted by Rhoads and Dick,[1] twenty-nine roentgenographically negative pulpless teeth were extracted. The root-ends were "snipped" off, ground in a sterile mortar and cultured. It was found that 51.7 per cent of all the pulpless teeth showed greater bacterial growth than the control teeth, which had vital pulps. Rosenow,[2] in 1919, performed a similar group of experiments and then injected 5 cc. of a broth culture, made from the infected tooth, into an animal intravenously. Two or three days later, the animal was killed, and it was found that, from almost all of the tooth cultures, it was possible to produce infection in the injected animal. Eusterman[3] examined the dental films of 900 patients at the Mayo Clinic and found 7 per cent of all teeth to be pulpless. Eighty per cent of the pulpless teeth showed apical lesions, although some rarefaction areas were minute. Blayney, Coolidge, Hatton and Kronfeld[4] studied the histopathologic reaction at the apices of pulpless teeth, and all reported that, in the absence of infection, cementum is deposited upon the root and in the unfilled part of the canal. None of these methods of studying the condition of pulpless tissue has proved entirely satisfactory.

The concept prevailing among many practitioners that every radiolucent area about the apical region of a tooth con-

*Summary of a thesis submitted in partial fulfilment of the requirements of the graduate school of the University of Illinois for the degree of master of science in dental pathology and therapeutics.

stitutes a definite indication of infection is wholly unsupported by factual evidence. Somner and Crowley[5] attempted to correlate the bacteriologic with the roentgenographic findings in pulp-involved teeth and reached the following conclusions:

1. It has been pointed out that many types of bone characteristics which appear to be abnormal may be normal for certain individuals or for certain specific regions and so may not necessarily indicate the presence of infection.

2. As bacteriologic findings do not wholly confirm the roentgenographic appearance of periapical lesions, too much emphasis should not be placed on the roentgenogram as a diagnostic agent in condemning questionable teeth.

Gutziet,[6] in 1937, described a method of applying short wave diathermy to the apical periodontal tissue which he believed permitted differentiation between foci that cause disease in other tissues and those that do not. The heat produced by the short waves was applied to the roots of the tooth to be tested by means of a special set of electrodes. One of these electrodes was applied on the lingual surface of the tooth and the other on the labial or buccal surface. Gutziet tested normal and pulpless teeth, with the following conclusions: (1) short wave irradiation of healthy teeth with living pulps does not cause an acceleration of the sedimentation rate of the red blood cells, and (2) similar irradiation of some of the teeth that have granulomas or necrotic pulps does cause an acceleration of the sedimentation rate. Gutziet studied only five cases, but his results were interesting enough to warrant further study.

The purpose of my studies was fourfold: (1) to determine the effect of short wave diathermy on the apical periodontal tissue of teeth that were normal clinically and roentgenographically, and of pulpless teeth, with or without roentgenographic evidence of periapical disturbance (2) to correlate the relationship of the blood sedimentation rate and the roentgenographic condition of the tooth; (3) to correlate the blood sedimentation test with the histologic examination, and (4) to correlate the histologic evidence and the roentgenographic appearance of infection. It was hoped that this test would indicate the absence or presence of infection in the apical periodontal tissues.

BLOOD SEDIMENTATION RATE

The blood sedimentation rate, a nonspecific reaction, can be compared to the temperature range, pulse rate and blood count, because it gives information of general character. It is a measure of the presence and intensity of the morbid processes within the body. In localized acute inflammatory conditions, variation in the sedimentation rate is dependent on the nature and severity of the morbid process. The rate varies with the extent and nature of the infection.

SHORT WAVE INDUCTOTHERMY

Short wave diathermy was used to heat apical periodontal tissues to permit study of the effects of possible foci of infection upon the blood sedimentation rate. Short wave diathermy is an electrical current which produces heat in a conductor in proportion to the resistance encountered, and it is designated in terms of wave lengths from 3 to 30 meters and frequencies in the range from 10 to 100 megacycles. The wave length is determined by dividing a constant by the frequency at which the current oscillates. Worster[10] states that the apparatus which produces short wave current is made up of a transformer and radio broadcasting tubes. The transformer raises the 100 volt alternating current to at least 1,000 volts. This can be delivered directly to the radio tubes, which act both as a rectifier and as an oscillator. The radio tubes are evacuated glass bulbs containing grid and plate. The filament is

heated to incandescence by the low voltage alternating current of the same frequency as the input current. A high direct current voltage of 1,000 volts is applied between the plate and filament, the plate being made the positive and the filament the negative.

To determine the amount of heat produced in the dielectric, which is the body or any part of the body, two factors must be considered: the capacity current and the dielectric constant. The dielectric constant is the specific conductivity of the various tissues of the human and animal body. Schereschewsky[17] has shown that the dielectric constant of different tissues is practically equal to that of water, 76-78, with the exception of fat, which has the low value of 13.6. The capacity current is produced by the wattage and the distance between the condenser plates. As a general rule, the higher the frequency, the greater the penetration and concentration of the heat. The frequency at which the maximum heating takes place is related to the electrical conductivity and the dielectric constant by the following formula[18]:

$$\text{frequency} = \frac{2 \times \text{specific conductivity}}{\text{dielectric constant}}$$

The method of producing heat in the dielectric is not definitely understood. There have been many theories, but the one which appears to be most applicable is known as dielectric hysteresis. With this method, it is taken for granted that each molecule of the body, called a dipole, contains a positive and negative charge in its respective sides. As the current changes its polarity, a similar alteration takes place in the condenser plates. With this change, each dipole of the dielectric is supposed to change with it, only in the opposite direction. When one of the plates is positive, the negative side of the dipole is drawn toward it, and when the plate is negative, the positive is attracted. In this polarity change, the dipole bends back and forth, and the friction of this creates heat directly in the tissues.

Thus, it was believed that if a diathermic current were passed through the apical periodontal tissue and the temperature of the tissues increased, the focus of infection in that area would be provoked and toxins located there would be liberated into the blood stream. Cignolini and Oliveri[19] observed the interdigital web, mesenteries and tongue of frogs in the field of short wave diathermy. According to the current employed, they found (a) with small doses, an acceleration of the blood flow and a slight dilation of the capillaries; (b), with moderate doses, a constriction of the capillaries; (c), with strong irradiation, an ischemia with almost complete occlusion of the capillaries, and (d), with a great output, a transient vasoconstriction, then vasodilation and acceleration of the blood and, finally, a slackening, ending in stasis. Cignolini and Oliveri conclude that capillaries respond to short wave exposures just as they do to heat.

MATERIALS AND METHODS

For this study, a model "b" inductotherm was obtained from the General Electric X-Ray Corporation. A special set of electrodes were also made by the engineers of General Electric (Fig. 1), so designed that they can be applied to the gum tissue over the apex of the tooth to be tested. One of the electrodes is placed on the buccal or labial surface of the gum; the other, on the lingual surface. The electrodes are designed as to size and position so that the heat produced by the inductothermy will pass through the region of the apex of the tooth. Heat can be felt only at one of the two electrodes, and this electrode was always placed on the lingual surface to prevent heating of the lips and cheeks. The amount of heat produced by this model inductothermy can be controlled by adjusting the resistor on top of the

machine. To improve the control of heat produced by the special electrodes, an additional coil has been added to the "surgical" end of the machine. This coil consists of 10 turns of 3 inches of one-quarter inch (outer diameter) copper tubing, taped at eight points to give four different intensities of heat.

LABORATORY EXPERIMENTS

To determine the efficiency of this method, various experiments were performed. The first group of experiments were made on dogs. The dogs were first anesthetized with pentobarbital sodium, and the electrodes were applied to the dog's jaw, one electrode on the labial or buccal surface; the other, on the lingual surface. Various doses of diathermy were applied for a few minutes and the temperature increase determined with a calibrated thermocouple. Results indicated that the temperature of the area could be raised as much as eight degrees with a special electrode, and the increased temperature was localized in the area heated. However, it was believed that these measurements were not accurate. In determining the temperature of a tissue heated by short wave diathermy, the customary procedure is to shut off the diathermy current and insert the thermocouple needle in the heated tissue. In the interval between shutting off the current and final equilibrium of the galvanometer, a considerable fall in temperature probably occurs.

Fig. 1.—Application of electrodes, showing position of each about apex of tooth to be tested.

In another animal, diathermy of a dose to increase the temperature only a few degrees was applied for fifteen minutes. Immediately after the heating period, the dog was killed and the jaw removed and placed in a solution of 10 per cent formalin for fixation. The areas of the jaws to which the heat had been applied were sectioned for histologic study. Where the temperature was raised between two and three degrees Fahrenheit above the normal temperature of the dog, the histologic sections showed large areas in which the blood vessels were markedly enlarged and filled with many red blood cells. All of the capillaries were engorged, but there were no signs of any burning of tissue. (Fig. 2.) The results of the animal experiments showed that, with the application of heat to the tissue, two to three degrees above the normal temperature, an increased amount of blood flowed through the blood vessels in the area heated.

Fig. 2.—Tissue around tooth of dog to which inductothermy was applied just before dog was killed. The tissue is not burned. The capillaries are enlarged.

CLINICAL STUDY

Studies of the effects of short wave penetration on periapical tissues were made in forty-two patients from the clinics of the University of Illinois, College of Dentistry. Roentgenograms of the teeth to be tested were obtained in all cases, and the patient's age, sex and general physical condition were noted. The physical histories of those patients who were referred from the Research Hospital were studied. The cases were divided into two groups: first, those whose teeth were normal clinically and roentgenographically; second, those patients who had pulpless teeth, with or without roentgenographic evidence of periapical disturbance, and whose teeth were condemned for extraction for reasons of health or for restorative purposes.

Tests were carried out in the following manner in all cases. Before the diathermy or inductothermy, which are one and the same, was applied, approximately 5 cc. of blood was drawn from the cubital vein by means of a dry syringe and ejected into a small bottle containing 4 mg. of solid potassium oxalate and 6 mg. of solid ammonium oxalate. The blood and anticoagulant were well mixed. The blood sedimentation rate was determined by the method described by Wintrobe. A Wintrobe hematocrit was filled to the 10 cm. mark and the tube placed in a vertical position. This was allowed to remain for exactly one hour and a reading was then made. The hematocrit was then centrifuged until all of the red blood cells were packed down tightly and the volume of the packed red cells was determined. In accordance with the chart devised by Wintrobe, the rate of sedimentation was corrected to the normal volume of packed red blood cells for women or for men. The need for correction is based upon the fact that the number of red blood cells varies and, to standardize the rate, the Wintrobe chart corrects the volume of cells to the equivalent of 5,000,000 red blood cells for men and 4,500,000 red blood cells for women.

The subject then was seated in a dental chair and the electrodes applied to the gum of the tooth to be tested and held in position for fifteen minutes. The electrodes were applied so that the hot electrode was placed on the lingual side of the gum. The dosage of heat was controlled primarily by the patient's tolerance. An additional check was made by placing a thermometer in the axilla and noting the temperature. At no time was the axillary temperature allowed to rise more than two degrees, because the tolerance varies so greatly that burns can be produced in the mouth of some patients before they object to the heat. Two hours after application of the heat, blood was again removed from the vein and a second sedimentation rate test made. The results of the two tests were compared and recorded. The results were classified in three groups: (1) no change, (2) decreased and (3) increased rate. Differences of more than 1 mm. were considered as significant.

The first or normal control group consisted of eight subjects, all of whom had teeth that were normal both clinically and roentgenographically. Two patients were women and six were men. The ages varied from 21 to 55 years. Five cubic centimeters of blood was drawn from the vein and the preliminary sedimentation rate test was made. The electrodes were applied to the normal teeth and the machine was turned on for fifteen minutes. Both anterior and posterior teeth were tested. These patients rested for two hours after diathermy and then a second sedimentation rate test was made. The two tests were compared and the results noted. Five patients (62.5 per cent) were found to have no change in sedimentation rate and three patients (37.5 per cent), decreased sedimentation rate. None of the normal patients had an increased sedimentation rate.

A total of thirty-four cases comprised the second group. All these patients had pulpless teeth, with or without roentgenographic evidence of periapical disturbance. The ages in this group varied from 21 to 62 years. Twelve of the patients were men and twenty-two women. Five cubic centimeters of blood was drawn from the vein and the preliminary sedimentation rate test made. Two tests were compared and the results noted. Twenty-one patients were found to have an increased sedimentation rate and four a decreased sedimentation rate. In nine, there was no change. In addition, with this group of patients, attempts were made to have the teeth extracted

TABULATION OF PULPLESS CASES

	Cases	Increased Sedimentation	Decreased Sedimentation	No Change in Sedimentation
Total	34	21	4	9
		62%	12%	26%
X-ray negative	9	3	3	3
		33%	33%	33%
X-ray positive	25	19	1	5
		76%	4%	20%
Histologically negative	1	0	0	1
		0%	0%	100%
Histologically positive	22	13	4	5
		59%	18%	22%

one week after the test. The teeth were extracted by the oral surgery department of the University of Illinois, College of Dentistry, and as much as possible of the surrounding tissue was obtained. Immediately after the extraction, the teeth were placed in 10 per cent formalin for fixation. The teeth were then decalcified and embedded in celloidin, and serial sections were made. The individual sections were 12 micra thick and were stained with hematoxylin and eosin. These sections were studied and the histopathologic appearance was noted.

Two of these cases were roentgenographically indefinite.

Correlation of the findings give the following results:

1. Twenty-three of the thirty-four cases in group two were sectioned histologically after inductothermy. Of these, twenty-two showed inflammatory reaction and one tooth appeared to be normal histologically.

2. Roentgenographically, twenty-five teeth were positive and showed radiolucent areas about the root. Nine teeth appeared to be normal, with an undisturbed lamina dura and periodontal membrane and no radiolucent areas.

3. Analysis of the results shows that thirteen cases were histologically positive and had an increased sedimentation rate, and that eleven of these cases presented roentgenographic signs of infection and two did not.

4. Four cases were histologically positive and had decreased sedimentation rates. Three of these cases showed no signs of infection roentgenographically and one case did.

5. Five cases were histologically positive and showed no changes in the blood sedimentation. All were roentgenographically positive.

6. A study of the relationship of the x-ray picture to the appearance of the histologic section showed that (a) sixteen teeth were x-ray positive and histologically positive; (b) no teeth were x-ray negative and histologically negative; (c) six teeth were x-ray negative and histologically positive, and (d) one tooth was x-ray positive and histologically negative.

SUMMARY

1. Short wave inductothermy, when applied to the jaws of animals, heated the tissues two or three degrees above normal and caused an increased flow of blood to the region heated.

2. Forty-two patients were studied to determine the effects of inductothermy

on focal infections as evidenced by changes in the blood sedimentation rate. Of these, in 62 per cent of cases, the teeth with vital pulps showed no change in the blood sedimentation. There was an unexplained decrease in the blood sedimentation rate in 38 per cent.

3. Of the patients with pulpless teeth, 62 per cent, (twenty-one cases) showed an increase in blood sedimentation rate, 25 per cent showed no change and 12 per cent showed a decrease.

4. Of the twenty-one cases that had increased sedimentation rate, nineteen were also x-ray positive and thirteen histologically positive.

5. Twenty-three cases were studied roentgenographically and histologically. Of these, sixteen cases were positive both roentgenographically and histologically, and of the remaining cases six were histologically and x-ray negative and one was histologically negative and x-ray positive.

6. Results of this study indicate that while this method may have some merit, it is not accurate, simple or standardized sufficiently to justify its adoption as an aid in locating periapical infection. It may be an important indication as to the toxicity of the periapical infection.

7. This study indicates the need for a further and more detailed investigation of the problem, employing a greater number of cases of both normal and infected teeth. There is the possibility that those teeth that are associated with increased sedimentation rates are the ones that are actually foci of infection, and that those teeth that are not associated with an increased sedimentation rate, in spite of their roentgenographic and histologic evidence of inflammatory reactions, have no effect upon the general health of the patient.

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AN UNUSUAL CASE OF HYPOCALCIFIED ENAMEL*

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INTRODUCTION

THE recent reviews of Sarnat and Schour[1, 2] have admirably summarized our present knowledge of enamel hypoplasia. These authors have emphasized the complexity of the condition, indicating that the origin is unknown in more than 50 per cent of cases. They conclude that enamel hypoplasia is a dental manifestation of a constitutional disturbance which affects the enamel of the deciduous and permanent teeth during the period of calcification. Their data indicate that approximately two-thirds of the cases occur during infancy or from birth to the end of the first year. Most of the remaining one-third of the cases occur in early childhood or from the thirteenth to the thirty-fourth month. Less than 2 per cent have their origin in the late childhood period.

Most of the material reviewed by them is concerned with enamel hypoplasia involving a limited number of teeth. Generalized hypoplasia of the enamel of all the teeth is apparently an extremely rare entity. For this reason, the following case is reported in great detail. It is hoped that this procedure may lead to the recognition of common features in other cases and this in turn may help to improve our understanding of the mechanism responsible for the condition.

REPORT OF CASE

History.—C. M., a girl, aged 17½ years, referred by a private practitioner to the Tufts College Dental Clinic in the latter part of October 1942, complained chiefly at that time of inability to secure employment because of blackened and disfigured teeth.

Questioning indicated that she was an only child, born of Italian-American parents in Cambridge, Mass., and had resided in or around Boston during her entire life. Available information indicated that her birth was normal and that she was breast fed for twenty months. At about 9 months of age, she had a severe attack of whoopingcough, which persisted for about ten weeks before responding to the third in a series of unidentified injections. The illness was characterized by the continuous presence of an elevated temperature.

During this period and the one immediately following, it was recalled that eruption of the teeth was accelerated. In the words of the parent, "they seemed to come up all at the same time." The family reported that, during this period, the gum tissue became very much inflamed and the services of a dentist were sought. The dentist was reputed to have treated the condition by incision of the affected part all along the gum margin of the upper and lower teeth. The treatment was not particularly successful. Both the family and the dentist stated that the deciduous teeth were devoid of enamel and the crowns of the teeth were pitted and stained. The deciduous teeth were described as being black and extremely brittle.

The childhood and adolescent period was apparently uneventful. There is a record of measles at 6 and mumps at 9 years. The tonsils were removed at 14 years of age. At the time of the initial examination, the patient was very conscious of her affliction, attempting to hold her hand over her mouth when speaking or eating. Because of her condition, she had existed on a soft diet from infancy.

Since the case was unusual, the familial

*From Tufts College Dental School.

history was carefully solicited. It was learned that the mother and father were first cousins. The teeth of both parents were found to be normal, as was the dentition of the thirty near relatives that could be contacted, including aunts, uncles, cousins and one grandparent. Incidentally, the grandparent was 75 years old and had twenty-six teeth, none of which was carious.

Examination.—The patient was well nourished and apparently healthy. She proved to be above average intelligence and extremely cooperative. A full mouth x-ray picture was made (Fig. 1) and models were secured for study. Careful examination of the mouth revealed that, with the exception of a few spots, the enamel of all the teeth was missing. Extensive questioning failed to determine the extent of the enamel at the time that the teeth were erupted. The crowns were stained a deep brown and showed generalized caries. The posterior teeth showed deep occlusion. The bite in the anterior region was slightly open and the teeth were irregular. (Fig. 2.) A marginal gingivitis was present throughout the mouth. Roentgenograms indicated that the alveolar bone and the root formation were essentially normal and that the third molars were unerupted. There was evidence of pathosis at the apex of the lower right central incisor. Several radiolucent areas could be detected in the supporting bone. The most prominent of these areas was between the lower first lateral incisor and cuspid, the roots of these teeth seeming to be displaced laterally.

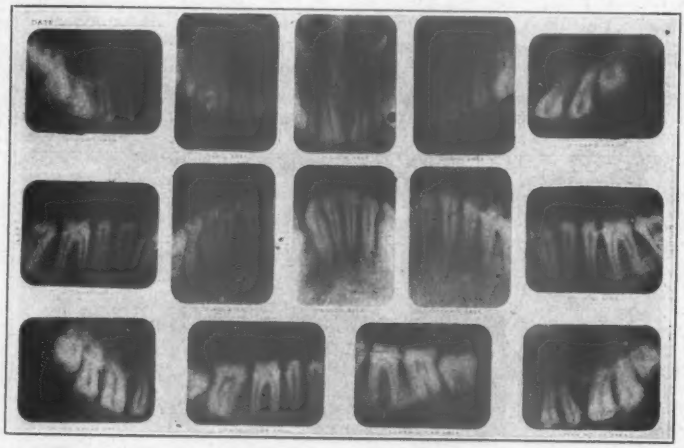

Fig. 1.—Full-mouth roentgenograms before treatment.

Fig. 2.—Teeth before treatment, anterior view.

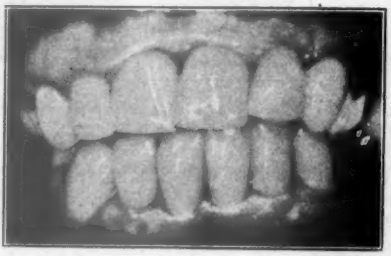

Fig. 3.—Completed case, anterior view.

Although the past physical record of the

patent was essentially negative, it was hoped that a complete medical checkup might reveal something of interest. Accordingly the patient was given a complete physical examination, the results of which were inconclusive. Likewise, x-ray pictures of the skull and long bones showed no abnormalities. The serologic examinations gave no clues as to the etiology of the disease. The serum calcium was 11 mg. per hundred cubic centimeters, and the inorganic phosphate 3.4 mg. The alkaline phosphatase was 2.69 units (normal range 1-4 units); acid phosphatase 4.4 units (normal range 1-4 units). The Hinton test for syphilis was negative.

Treatment.—After consultation, it was decided to attempt to restore, as far as possible, the normal anatomic structure and occlusion of the teeth. Since appearance was of prime consideration, it was decided to jacket the anterior teeth and the bicuspids and place full cast crowns on the molars. Because of the loss of the enamel on the surfaces of the postcanine teeth and the resultant deep occlusion, it was necessary to open the bite. The extent to which this could be accomplished was limited by (1) the fatigue position and (2) the open bite in the incisor region. The latter condition, plus the bite raising, necessitated an increase in vertical dimensions of the anterior replacement. It was decided to treat the root canal and place a cast core on the lower right central incisor. The third molars were marked for extraction.

After the new maxillary-mandibular relationship had been established with a bite-rim, the eight upper and lower molars were prepared for full cast gold crowns. The finished crowns were cemented in position at the same time. Next, the upper bicuspids were restored with acrylic jackets and the lower bicuspids with porcelain crowns. It was hoped that this procedure would permit a more normal occlusal attrition. For the same reason, the upper molars were cast in soft gold and the lower molars in hard gold. Shoulder preparations were used throughout.

The upper and lower front teeth were restored with porcelain jackets. Since the upper right lateral incisor was dislocated at an angle of about 45 degrees in an axial direction, it was difficult to bring it into a fairly normal position. Because of this, it was necessary to partially overlap the contacting upper right central incisor. The completed case is shown in Figure 3. Roentgenograms in Figure 4 show the completed case.

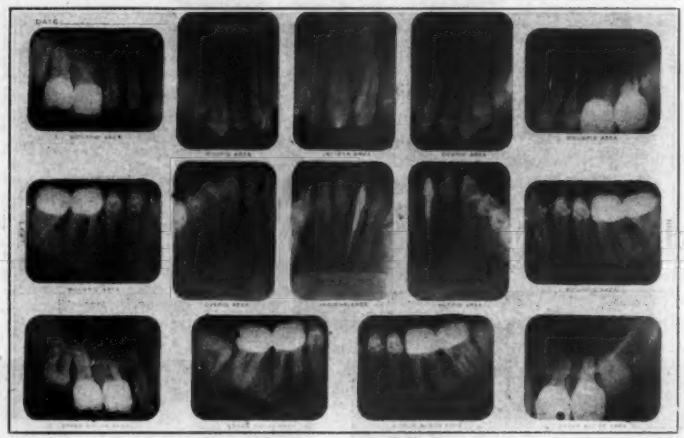

Fig. 4.—Full-mouth roentgenograms after treatment.

Comment.—It is interesting to note that although the teeth with one exception were vital (Burton electric pulp-test), it was not necessary to administer anesthesia in any form to facilitate the required operative procedure. Postoperatively, the patient experienced little difficulty in adapting herself to the new occlusal relationship. After twelve

months, the result is judged to be completely satisfactory. The marginal gingivitis has completely disappeared, and the pathologic area at the apex of the lower right incisor has healed. The chewing pressure in the molar region, measured with the Haber gnathadynamometer before treatment was instituted, was between 18 and 20 pounds. Today, it is between 28 and 30 pounds. Finally, it can be said that the mental attitude of the patient is vastly improved. She is now gainfully employed and has widened her social contacts.

I made all of the preparations and did the laboratory work. The case was completed within twenty-eight days.

study. Tissue sections were prepared by grinding and decalcification.* The following are the pertinent microscopic findings:

Decalcified sections: The enamel matrix was not removed by the decalcifying agent. It remained intact and the unaltered rod spaces were clearly visible. Along the entire proximal margin of one of the teeth, there was a narrow pale-staining zone of enamel which conformed to the contour of the crown and which was demarcated from the unaltered enamel matrix by a diffuse darkly staining line. (Fig. 5.) The peripheral enamel zone presented a hazy, amorphous, slightly fragmented appearance. Outlines of enamel rod spaces were barely perceptible. A clearly

DATA ON ENAMEL SPECIMENS

Source	Clinical Appearance	Specific Gravity	Average Specific Gravity
Axial enamel	Firm, pigmented, glossy, slightly rippled (most normal in appearance)	2.11 2.19 2.18 2.18	2.165
Occlusal surface enamel	Pigmented, relatively soft, surface continuity broken	2.08 1.95	2.015
Cusp tip enamel	Crumbled, granular, fragmented	2.04 2.03 2.08 2.05	2.04
Coronal dentin		2.095	2.095

The extracted upper and lower third molars were prepared for histologic examination. It was hoped that detailed observation of these teeth might offer some information as to the etiology of the hypoplasia.

Laboratory Data.—Following is a report of Dr. Irving Glickman, of the department of oral pathology:

Four molars were received for microscopic

staining, non-cellular cuticle was observed along the enamel surface externally from this peripheral zone.

An area of carious penetration and destruction was present on the occlusal surface of one of the molars.

Ground sections: There was an obtuse angular depression in the surface continuity of the enamel of one of the teeth. (Fig. 6.) Immediately below the depression, the enamel was markedly granular and devoid of rod spaces. The granular zone extended pulpally for approximately one-third of the thickness of the enamel. At this point, it apparently blended with the unaltered enamel structure.

The coronal dentin presented an unusually

*Decalcification technic: The teeth were fixed in 10 per cent formaldehyde, embedded in cellodin, decalcified in formic acid solution (45 per cent formic acid C.P., and 20 per cent sodium citrate, equal parts), run up in butyl alcohol series, and then embedded in paraffin.

large number of prominent interglobular spaces.

In order to supplement the microscopic findings, the specific gravity of the enamel and the dentin was determined by Dr. Deakins, of the Harvard School of Dental Medicine. The enamel specimens were grouped according to their clinical appearance as shown in the accompanying table.

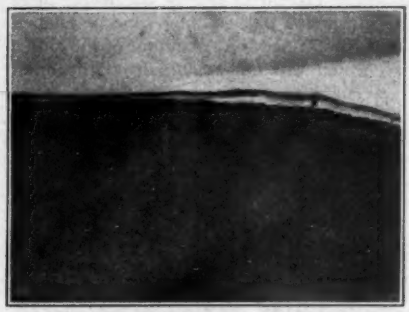

Fig. 5.—Decalcified section, upper left third molar, showing amorphous, slightly fragmented peripheral enamel zone along proximal surface. (Hematoxylin and eosin stain.)

The average values for normal enamel and dentin determined by previous studies[2, 3] were for enamel 2.945; for coronal dentin, 2.112.

Conclusions.—The following conclusions may be derived from a correlation of the microscopic and physical findings:

With the exception of an isolated zone of hypoplasia (Fig. 6), the enamel matrix was well formed throughout. Nevertheless, it was apparently incompletely calcified in the course of development. The insolubility of the enamel matrix in the decalcifying solution and the low specific gravity of the intact axial enamel support this view.

It is believed that decalcification of external origin resulted in peripheral fragmentation and granularity of the enamel. Support for this belief can be seen in the finding that the specific gravity of the granular enamel was even lower than that of the subnormal intact axial enamel. This suggests that the original low mineral content which resulted from incomplete calcification in the course of development was secondarily reduced by decalcification of external origin.

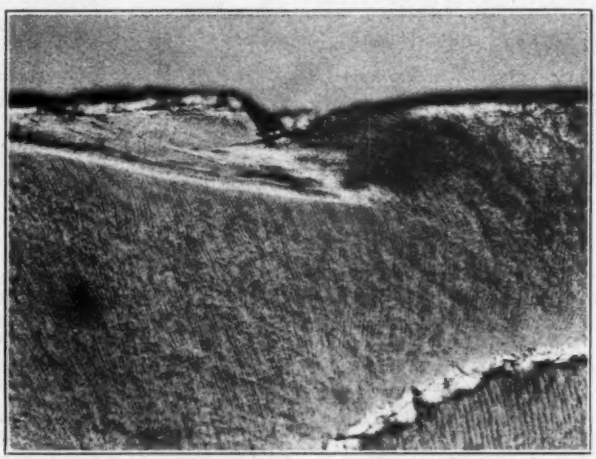

Fig. 6.—Ground section, upper right third molar, impacted, showing angular hypoplastic zone on proximal surface.

The generalized chalky disintegration of the enamel observed clinically in all of the teeth in this patient probably resulted from a combination of two conditions: (1) insuf-

ficient calcification in the course of development and (2) decalcification of an exogenous origin.

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ORAL MANIFESTATIONS OF HISTOPLASMOSIS

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HISTOPLASMOSIS, a fatal fungous disease, was discovered by T. S. Darling[1] in 1905. Seventy cases have been recognized and described to date. From a dental standpoint, histoplasmosis is important because 20 per cent of the persons infected had oral lesions. A review of these cases is presented in order to bring the disease to the attention of dentists. It is hoped that knowledge of the disease and its oral manifestations may serve as an aid to diagnosis.

Histoplasmosis is characterized clinically by a low grade, irregular pyrexia, splenomegaly, hepatomegaly and lymphadenopathy. Anemia and leukopenia are observed in most cases. Ulcerated granulomatous lesions of the skin, oral mucous membranes, pharynx and gastrointestinal tract occur. It is seldom that these organs and tissues are affected uniformly. The disease can be localized or systemic. It may involve any organ or organ system. Signs and symptoms encountered are referable to the organ or system involved. The disease may run an acute or a chronic course, lasting from a few weeks to many years. Regardless of the time interval involved, the fungus seems to have the ability to develop relentlessly at the expense of the host until death occurs.

From a dental standpoint, histoplasmosis should be an important entity because of its tendency to simulate known diseases, such as carcinoma, leukemia, noma, leishmaniasis, Hodgkin's disease, fusospirochetosis and others.

HISTORICAL REVIEW

In 1905, Darling went to the Panama Canal Zone to search for cases of visceral and cutaneous leishmaniasis. He found three patients with a disease similar to, but not identical with, it. A protozoan-like organism was observed in the tissues studied microscopically after necropsy. Darling named this organism *Histoplasma capsulatum*.[2, 3, 4] Rocha-Lima,[5] in 1912, called attention to its similarity to *Cryptococcus farciminosus (Blastomyces farciminosus)* and concluded that *Histoplasma capsulatum*, like the cryptococcus, was a fungus.

From 1906, when Darling first described histoplasmosis, until 1934, only nine cases were reported. In 1943, De Monbreun[6] cultivated the etiologic agent, the fungus *Histoplasma capsu-*

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latum, and proved its pathogenicity. Since that time, sixty-one cases have been reported or referred to in the literature. An increase in the reported incidence of a disease always raises the question as to whether there is an actual increase in the number of cases or whether it is being more generally diagnosed. Meleney[7] believes that the recent rapid increase in the number of cases observed is due to more general recognition of the disease, but that many cases are still unrecognized. Humphrey[8] believes that the disease is actually increasing in incidence.

REVIEW OF CASES OF DENTAL INTEREST

Four of the outstanding cases will be reviewed in detail, while the others will be described more briefly.

The case presented by Moore and Jorstad[9] is of particular interest since the diagnosis was made entirely from the oral lesions.

REPORT OF CASES

A white man, aged 67, seen at the hospital on August 17, 1942, complained of a "sore" on the roof of his mouth, noticed two months previously and gradually increasing in size. There was no pain or tenderness in that area. A biopsy specimen was taken from the right upper alveolar process. The clinical impression at that time was carcinoma of the buccal mucosa.

Microscopic examination of the tissue revealed a markedly hyperplastic mucous membrane with an ulcer in one section of the specimen. Beneath this ulcer was a mass of dark staining cells. In this mass were young fibroblasts, epithelioid cells, polymorphonuclear leukocytes, lymphocytes, eosinophiles and numerous large macrophages. In the cytoplasm of these macrophages and also extracellularly, there were many small organisms characterized by a central, dark-staining body surrounded by a clear capsule. These organisms were about 2 to 4 microns in diameter. A diagnosis of histoplasmosis of the upper right alveolus was made.

August 25, 1942, the patient entered the hospital. Examination revealed an ulcer measuring 3 cm. in diameter on the lingual edge of the alveolar border, which extended along the right upper first, second and third molars. A slight discharge from the ulcerated lesion was noted. Physical examination revealed a palpable liver 2 fingers breadth below the costal margin. It was not tender. The spleen was palpable 4 fingers breadth below the costal margin. It was hard, and not tender.

August 26, under general anesthesia, the ulcerated area was removed by cautery with a 1-cm. margin of apparently normal tissue. The molars and premolars were removed and this area was also cauterized. The periosteum underlying this area was included in the removal. September 9, a piece of tissue removed from the lower gum was examined for the presence of *H. capsulatum.* No evidence of the organism was found. The patient left the hospital, but returned November 12. At this time, "There was an area of granular, fungating and ulcerative tissue which extended from the gingival border of the lower incisors, occupying the buccal sulcus, almost to the rim of the lower lip. The lower incisor teeth were loose, tender on pressure, dirty and carious. The area previously cauterized remained healed. The area of the roof of the mouth posterior to the left incisors was ulcerated." November 13, the involved area was destroyed with cautery under local anesthesia. The four lower incisors were extracted and the sockets cauterized. Pieces of tissue were removed for microscopic examination during the operation. The organisms of histoplasmosis were found in abundance in all specimens examined, including one from the antral mucosa. The patient left the hospital again and reentered in December 1942. During the intervening time, his mouth had become very painful. There was marked *fetor oris.* The patient died January 7, 1943. Permission for necropsy was not granted.

Simson and Barnetson,[10] in describing the first case reported from Africa, that of a white man aged 55, stated that the chief complaint was difficulty in swallowing. On physical examination, a small conical nodule was noticed on the dorsum of the tongue at the junction of the posterior and middle thirds. This lesion ulcerated and discharged a small amount of serous fluid for several weeks, after which several lesions occurred

on both surfaces of the tongue, on the upper and lower gums and on both lips. These lesions were raised, conical, red, moderately hard and painless and about three-eighths inch in diameter. They broke down to form shallow ulcers, one-fourth inch in diameter, with an erythematous border and a purulent base. Microscopic examination of material scraped from the ulcers showed intracellular and extracellular yeastlike bodies. Clinical examination revealed enlargement of the spleen, liver and cervical lymph nodes.

A biopsy of the lesion on the lip and tongue revealed the following histologic changes: The surface epithelium was thickened, with a slight excess and heaping up of keratin. The interpapillary processes were irregular in shape and extended into the corium. There was proliferation and infiltration of the reticulo-endothelial cells into the corium and parts of the underlying muscle. Occasionally, they were arranged in "follicle formation superficially resembling tubercle follicles." A few giant cells of the Langhans type were present. Plasma cells, eosinophiles and small lymphocytes were found scattered among the reticulo-endothelial cells. Large numbers of these reticulo-endothelial cells were packed with the typical yeast forms of *Histoplasma capsulatum*. The patient refused treatment, left the hospital and has not returned.

In the case described by Brown, Havens and Magath,[11] the patient was admitted to the Mayo Clinic in June 1940, with a chief complaint of sore throat, which had persisted since the extraction of several lower teeth two years previously. The patient was a white man, aged 48. In January 1939, the soreness of his throat became more acute and, at this time, a cough, fever and loss of weight were noted. The severity of the symptoms gradually increased and, in April 1939, he was given a course of x-ray treatment, which gave some temporary relief. In August 1939, several specimens were removed from the throat for biopsy. The pathologist reported non-specific granulomatous tissue.

Physical examination revealed that the patient was emaciated with no signs of tuberculosis or other wasting disease. His weight had decreased from 217 to 109 pounds in the past two years. The entire soft palate had a fine granular grayish appearance and was studded with numerous small hemorrhagic patches. The uvula was irregularly shaped and nodular. General adenopathy was not present. The liver and spleen were not palpable. At this time, June 1940, a biopsy of the pharynx was made. The pathologist again reported "indeterminate granuloma." Several days later, another report was made, this time of leishmaniasis. After further study, a final pathologic report was made and the diagnosis of histoplasmosis was established. The patient died in July 1940.

At necropsy, a pinkish gray granular ulcerating lesion involving the hard and soft palate, with extension onto the uvula and pillars of the tonsils, was found. The bronchi, mesenteric nodes, peritoneal mucosa, small intestine and large intestine were involved in an ulcerative process. The diagnosis of histoplasmosis was confirmed.

Palmer, Almosch and Shaffer[12] reported a case of mucocutaneous histoplasmosis in a 45-year-old white man. He was admitted to the hospital with a chief complaint of severe hoarseness, pain in the throat and pain on swallowing of five months' duration. Four months before his admission, an ulcer developed on the left side of the upper lip. In the week before admission, he had noticed similar ulcers on the penis and about the anus. Examination revealed a deeply punched-out destructive lesion covered by a dense crust. There was no induration or inflammatory reaction. There were numerous superficial ulcerations of the mucous membrane of the mouth, especially on the tongue and the palate. *Histoplasma capsulatum* was recovered from the lesions on the lip and in the perianal region. Despite chemotherapy, which consisted of local applications of potassium permanganate, gentian violet, 2 per cent thymol in olive oil and sulfanilamide dust powder, and intravenous administration of fluids, high vitamin dosage, transfusions and stibamine glucoside and sulfathiazole therapy, the patient died about four months after the diagnosis was made.

The necropsy report failed to confirm the diagnosis, since the organism could not be found in any of the internal organs either on microscopic inspection or by cultural methods.

Agress and Gray[13] reported the case of a 7-months old boy whose chief symptom

was recurrent epistaxis of one month's duration and slowly healing sores about the lips. These lesions were suggestive of rhagades. A small ulcer appeared in the buccal mucosa, but there was no cervical adenopathy.

Although the serologic examination of both parents and the infant was negative, a diagnosis of congenital syphilis was made. The patient's health declined, and he died nineteen days after admission. The final diagnosis of histoplasmosis was made at necropsy.

Hansmann and Schenken[14] observed one buccal lesion, which was followed a year later by several small granular, ulcerated lesions on a thickened oral mucous membrane, in a man aged 43 with histoplasmosis.

Humphrey[8] reported a case in an 18 year old boy who had a number of bullous lesions on the face and body. A stomatitis developed from which Vincent's organisms were recovered. After five days of sulfanilamide therapy, these lesions subsided. Seven weeks later, however, some ulceration of the roof of the mouth still existed, and large masses of palpable matted glands were present on each side of the neck. Diagnosis of histoplasmosis was made at necropsy.

The case of Williams and Cromartis[15] was that of a man aged 65 with lesions of the soft palate, pharynx and nasal mucosa.

Negroni's[16] case in a man aged 40 with vegetations and ulcerations of the soft palate and nares proved to be histoplasmosis.

Van Pernis, Benson and Hollinger[17] reported the case of a man aged 65 with ulceration in the premolar region and involvement of the tongue, true and false cords, larynx, tonsils and pyriform sinuses.

Moore and Jorstad[9] refer to five other cases of histoplasmosis, which have not yet been reported, in which oral lesions were outstanding findings.

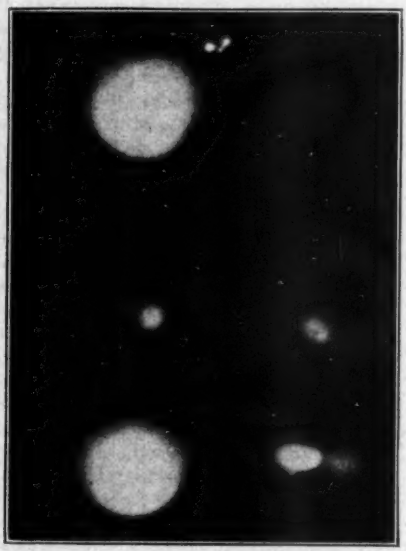

Fig. 1.—Typical fluffy white colony of Histoplasma capsulatum grown on dextrose agar at room temperature. (×2.)

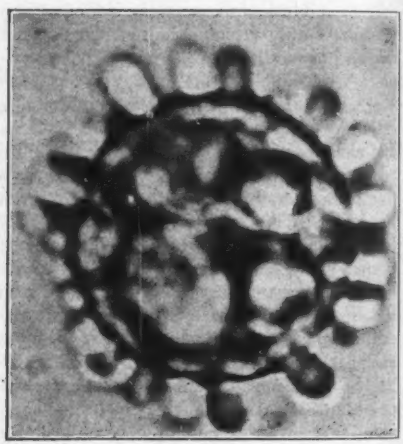

Fig. 2.—Typical tuberculated chlamydospore. (×700.)

THE ORGANISM

Histoplasma capsulatum is readily grown on such routine media as Sabouraud's glucose, maltose agar or corn meal agar. Under aerobic conditions, at room temperature, the organism will develop in about ten days. This is the typical mycelial form (Fig. 1), showing septate hyphae, from 1 to 3 microns in diameter. The characteristic spore (Fig. 2), a thick walled chlamydospore, develops on the aerial mycelium. It is at first smooth walled, but, in older cultures, the typical

tuberculated spores are seen. If the organisms are placed on sealed blood agar slants, the yeast form of the histoplasma will develop. Microscopically, these appear as thin-walled oval bodies about 2 by 4 microns, producing a single bud at the pointed end. A clear halo is visible around these cells.

PATHOLOGY

The liver, spleen, lungs, lymphoid tissue, bone marrow and blood stream are invaded by the organism to a greater or less degree. The reaction to the presence of the organism is a proliferation of the reticulo-endothelial system, the cells of which phagocytose the organisms. The macroscopic pathology is quite variable. Meleney,[7] in his review, stated that most cases show gray or white nodules or extensive areas of necrosis in one or more organs. Lung abscesses, enlargement of the liver and spleen, caseation of the adrenals, slight erosions or deep ulcers in the small or large intestine and ulcerations of the oral mucosa are common findings.

Microscopically, *Histoplasma capsulatum* is nearly always found in all of the above-mentioned lesions. The organisms are usually packed within the reticulo-endothelial cells. They are characteristically round or oval yeastlike cells, approximately from 1 to 4 microns in diameter, surrounded by a clear refractile non-staining capsule, with the nuclear material arranged near one pole of the organism. They have been observed in the bone marrow within large vacuolated endothelial cells, and have also been demonstrated in peripheral blood smears.[18] For more detailed macroscopic and microscopic pathology, the reader is referred to the literature, almost all cases reported having been presented in great detail.

DISCUSSION

Histoplasmosis of the oral cavity may involve any or all of the structures present. Nodular ulcerative or vegetative lesions on the tongue, lips, buccal mucosa and hard and soft palate are suggestive of histoplasmosis, whether one or all of the above-mentioned areas are involved. The buccal mucosa is most often involved by an ulcerative process, which spreads over the gums. These ulcers are usually covered with a gray or pinkish membrane, very similar to the membrane found in many ulcerative processes in the mouth.

Moore and Jorstad[9] reported a case in which the "clinical impression" was carcinoma of the buccal mucosa. The case of Agress and Gray[13] was diagnosed congenital syphilis because of the clinical appearance of the lesion, even though the serologic examination of both parents was negative. Palmer, Amolsch and Shaffer[12] reported an unusual case of histoplasmosis, "in which there was a manifestation heretofore not recorded, namely mucocutaneous involvement closely resembling mucocutaneous (American) leishmaniasis." Vincent's organisms have been recovered from the lesions of the case reported by Humphrey,[15] and the case was treated as Vincent's stomatitis. The fact that some cases showed cervical adenopathy,[15, 10] with enlargement of the spleen, is suggestive of Hodgkin's disease. In the differential diagnosis of carcinoma, of syphilis, of leishmaniasis, of Vincent's infection, of leukemia, of Hodgkin's disease and of other granulomatous or ulcerative processes in the mouth, histoplasmosis must be considered if we are to be of service in the early diagnosis and therefore ultimate control of this disease.

The key to diagnosis of a rare disease is found in the conscious realization of the possibility of its occurrence. Since there is no method of diagnosing histoplasmosis other than demonstration of the organisms in tissue cells or isolation of the organism on culture media, it is well to keep the possibility of histoplas-

mosis in mind whenever we are faced with a chronic granulomatous process in the mouth.

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THE JOURNAL *of the* AMERICAN DENTAL ASSOCIATION

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FEBRUARY 1 — *Editorial Department* — 1945

DENTAL PERSONNEL IN THE UNITED STATES

THE induction of more than one-third of the country's dentists into the various services during the war period has sharply emphasized the problems of dental personnel. The critical needs of both the military and the civilian population have, by and large, been met, but only through the extraordinary and sustained effort of dentists in and out of the services. This effort has been recognized universally as a valuable service to the nation, but it must not be used to obscure the fact that there is little, if any, reserve of dental manpower for further emergencies or for a large expansion of dental service.

The problems of dental personnel are among the thorniest to be solved before there can be an effective development of a program for dental health on a national scale. Morrey's[1] summary of available data on dental personnel points up many of these problems: (1) only in the past five or six years has the profession been able to increase its birth rate of new graduates to the point at which it exceeds the annual rate of death and retirement; (2) the distribution of dentists is spotty, and there are many areas, particularly rural ones, that are without sufficient personnel even to meet minimum standards of dental care; (3) the number of dental hygienists has not come close to matching the increase in the public's appreciation of dental services; (4) the gross income of the commercial dental laboratories has increased from 15,000,000 in 1920 to an estimated 125,000,000 in 1944, and the growth in technical personnel has matched this phenomenal growth in gross income.

1. See page 131 of this issue.

Even as isolated facts, these statements are significant. When they are set in the context of the problem to provide more and better dental service and for more people, they are important. They indicate the need for further study and as prompt action as possible in many quarters. The necessity for a vigorous program of vocational guidance on the part of the dental schools is obvious. Such a program should be directed, not at the mere multiplication of numbers in the dental schools, but also to attracting to the study of dentistry well-qualified students who might otherwise overlook the opportunities of the profession. A program to aid the returning dental officer in reestablishing himself in practice should be initiated by state and local dental societies. Information should be made available in each state on the need for additional dentists in certain areas so that this can be used as a basis of selection for those officers who desire to relocate their practices. The possibility of providing a subsidy to aid in this relocation and redistribution process should be carefully examined; and, finally, the profession at all levels of organized activity should work toward practical cooperation with all accessory groups who have a share in this great task of making dental care available to those who need it.

The problems of dental personnel are large and complex. There is no reason, however, for calling them insuperable. They will yield to careful study, intelligent planning, prudent experiment and informed action. Those are the tasks that lie immediately ahead.

VOLUNTARY PREPAYMENT PLANS IN DENTISTRY

The popularity and success of voluntary prepayment plans for hospital and medical care have marked an obvious and significant trend of the past decade in the expansion of health security in this country. More than fifteen million persons have voluntarily purchased hospital protection for themselves and their families in the thirty-two states that presently permit this type of program.

The possibility of applying the voluntary prepayment principle to dental service has long been studied and some valuable information has been gained from hospital and medical experience. There has been no conclusive evidence, however, based on the peculiar difficulties that surround any attempt to provide protection against such a common risk as dental disease. More evidence is needed before any judgment can be made as to whether voluntary prepayment plans for dentistry will, or can, meet with the popular approval and success of the hospital program.

An experiment designed to provide this essential information, which has been outlined by the Council on Dental Health of the American Dental Association,[1] will be put into operation as soon as proper sponsorship and sufficient funds to underwrite the project can be secured. It represents an attempt to meet a difficult problem scientifically and realistically.

There are many areas of the country which can provide ideal test conditions for this experimental program. Such areas, and the dentists and dental societies in them, should interest themselves in getting the program into operation without delay. If funds provide the initial obstacle, the possibility of securing them from the community, state or federal government under suitable conditions should be in-

vestigated. The voluntary prepayment program holds open such hopeful possibilities that there should be as little delay as possible in developing the experiment. Only from this method of trial and error can come the definite and final answers that should have a wholesome effect on dental health in this country.

A NATIONAL INSTITUTE FOR DENTAL RESEARCH

A BILL has been introduced into the U. S. Senate authorizing the appropriation of $1,730,000 for the erection of a national institute for dental research and for fostering dental research by public and private agencies. The National Institute for Dental Research would be a part of the National Institute of Health, Bethesda, Md., which is under the direction of the U. S. Public Health Service, Federal Security Agency. Similar institutes have already been authorized for research on cancer and tuberculosis and one has recently been proposed for mental hygiene. The dental institute will be erected and equipped at a cost of one million dollars, while the remainder of the funds authorized will be used to aid dental research by public and private agencies under the direction of the Surgeon General.

The bill was introduced by Sen. James M. Murray, Montana, who, on two previous occasions, sponsored bills for dental research. The present bill exceeds both of those efforts in scope and importance. Senator Murray has again given evidence of his substantial interest in providing a scientific approach to the problem of improved national dental health.

The new research bill is the translation into legislation of the first part of the program that has been declared repeatedly by the American Dental Association to be the basis of a successful, long-range program for dental health in this country. At the last annual meeting of the House of Delegates in Chicago, the Committee on Legislation was instructed to make all three points of the A.D.A. program effective through legislation. Dental health education and dental care, the remaining two points, will be treated in bills to be introduced at a later date.

The passage of this bill by Congress will insure a constructive beginning on a national program for dental health. Such legislation deserves the interest and constructive support of both the public and the profession.

COLOR

WITH this issue, THE JOURNAL makes its first use of color in the editorial section of the magazine. This medium has become commonplace to even the casual readers of popular publications, who have long been accustomed to multicolored illustrations. Its use in publications of a technical or scientific nature, however, has been limited, largely by the cost of reproduction. Under the new policy of the Board of Trustees which permits a rearrangement of the first part of the magazine, THE JOURNAL will undertake to employ color in order to present some scientific articles more graphically and more effectively. The comments of readers on this innovation will be appreciated.

COUNCIL ON DENTAL THERAPEUTICS

THE CURRENT STATUS OF VITAMIN D

THE Council on Dental Therapeutics of the American Dental Association has authorized publication of the following report.

DONALD A. WALLACE, *Secretary.*

It has been rather clearly demonstrated that vitamin D is of value in the formation of the hard dental structures, including the teeth themselves.

Evidence that this vitamin aids in the maintenance of the fully formed tooth or in the prevention or retardation of dental caries at any stage of its progress is not supported by observations that have passed beyond the controversial stage. This being the case, advertising asserting that the ingestion of milk or milk products, with or without vitamin D, will aid in the prevention or retardation of dental caries tends to jeopardize the reputation of these foods, since persons who use them liberally are not necessarily free from dental decay or other dental disease.

If it should be shown beyond reasonable doubt that vitamin D or products containing it are useful in maintaining normal tooth structure or in the prevention of dental disease, the Council on Dental Therapeutics will wish to foster as wide knowledge of such findings as possible. For the present, advertising claims for vitamin D as a factor in the prevention of tooth decay are not acceptable to the Council on Dental Therapeutics.

ACCEPTED DENTAL REMEDIES

The Council on Dental Therapeutics of the American Dental Association announces the inclusion of the following articles in the list of Accepted Dental Remedies:

ANESTHETICS—LOCAL*

Procaine HCl 2%, Epinephrine 1:50,000: Each cubic centimeter is stated to contain procaine hydrochloride, 0.02 Gm.; epinephrine 0.00002 Gm.; sodium chloride, 0.005 Gm.; sodium bisulfite, 0.0019 Gm., and distilled water. Marketed in tubes, approximately 2.3 cc.

Procaine HCl 2%, Epinephrine 1:25,000: Each cubic centimeter is stated to contain procaine hydrochloride, 0.02 Gm.; epinephrine, 0.00004 Gm.; sodium chloride, 0.005 Gm.; sodium bisulfite, 0.0019 Gm., and distilled water. Marketed in tubes, approximately 2.3 cc.

Procaine HCl 2%, Neo-Synephrine Hydrochloride 1:2,500: Each cubic centimeter is stated to contain procaine hydrochloride, 0.02 Gm.; neo-synephrine hydrochloride, 0.0004 Gm.; sodium chloride, 0.005 Gm.; sodium bisulfite, 0.0019 Gm., and distilled water. Marketed in tubes, approximately 2.3 cc.

Manufactured by S. F. Durst & Co., Philadelphia, Pa. Distributed by Stratford-Cookson Company, Philadelphia, Pa.

DENTIFRICES†

Style King Tooth Powder: Composition: See Sheffield Tooth Powder (A.D.R. Ed. 10, p. 103).

*A.D.R. Ed. 10, p. 39.
†A.D.R. Ed. 10, p. 95.

Manufactured by The Sheffield Company, New London, Conn.
Distributed by S. Buchsbaum & Co., Chicago, Ill.

Ain's Dental Cream: Composition: See Trade Brand Dental Cream (A.D.R. Ed. 10, p. 104).
Manufactured by the Trade Laboratories, Inc., Newark, N. J.
Distributed by Goodwear Products Co., New York, N. Y.

Admission of a product to the list of Accepted Dental Remedies does not imply a recommendation. It means that the product and the methods by which it was marketed at the time of consideration were not found to be in violation of the Council's published rules. Accepted products are reconsidered periodically. The files of the Council contain information on many drugs and dental cosmetics. All information is available, upon request, and inquiries are welcomed. A postal card inquiry will bring a prompt reply.

DONALD A. WALLACE, *Secretary.*

COUNCIL ON DENTAL EDUCATION

REPORT OF COMMITTEE ON POSTWAR EDUCATIONAL PLANNING

THE Council on Dental Education has appointed a committee on postwar planning consisting of J. Ben Robinson, chairman, William N. Hodgkin and Minor J. Terry. At a meeting of the Council held December 17, 1944, this committee submitted a report and a memorandum with special reference to postwar educational needs of the returning members of the armed forces. The following memorandum was adopted as representing the views of the Council:

The work of the Committee on Postwar Planning has been directed toward a study of the definitions of refresher, postgraduate and graduate courses, of the curriculums for these various courses, of the teaching time that should be devoted to them and of the physical and personnel facilities existing in the dental schools of the United States for providing such courses.

It appears that educators are not in full agreement on the definitions of the various types of advanced courses in dentistry. In the circumstance, it seems desirable for the committee to define refresher, postgraduate and graduate courses, not only for convenience in arranging educational programs for emergency needs, but also for use in planning long-range postgraduate programs for the profession. The committee suggests the following definitions as practicable for the use of dental schools in planning their advanced educational programs:

1. Refresher courses may be defined as short courses in clinical practice designed for the purpose of reviewing those clinical procedures learned, and of refreshing the performance skill developed by the student in the undergraduate curriculum. Such courses, while emphasizing instruction in new technics and improved clinical procedures, should include also a review of the literature and should require some organized formal study. They should refresh especially the students' use of basic procedures in the art of practice, but should be extended to include advanced instruction in new knowledge relating to the basic sciences and their applications. They may be offered in residence, or as extension projects. They do not earn credit toward a degree or lead to certification. They are *not* designed to equip the student for a specialty.

2. Postgraduate courses in dentistry may be defined as special courses pursued intensively and exhaustively by students who are graduates in dentistry. The outlines of such courses should incorporate planned instruction in background work, such as science laboratory and didactic classes, with the clinical work in a special field. Postgraduate courses do not lead to a degree, but may be rewarded by a certificate of proficiency and may lead to a specialty.

3. Graduate courses may be defined as

advanced courses planned for those especially prepared and qualified to do advanced study beyond the undergraduate level. The work done in such courses is of a charter acceptable to the graduate school of the university and leads to a degree upon completion of the requirements as specified by the graduate school. These requirements include individual research and the preparation of a thesis. The objects of such courses are to fit individuals for a specialty, for teaching or for research.

Your committee is not prepared to suggest the content or general range of curriculums for these various categories. The determination of the character and content of these various courses and the time to be assigned them as necessary to achieve their objectives require extended study before a decision concerning them can be reached. Therefore, the development of the curriculums should be left to the discretion of the respective dental schools, the faculties of which may organize the courses advantageously in terms of the foregoing definitions.

Earlier in its study, your committee sent a questionnaire to the deans of all the dental schools in a effort to determine to what extent the physical facilities now existing in dental schools may be made to provide for postwar educational demands. The following conclusions are based upon a careful survey of the replies received:

1. The great majority of the dental schools lack either suitable physical facilities or adequate teaching staffs to provide acceptable opportunities for the successful administration of refresher, postgraduate or graduate programs.

2. Only a very limited number of dental schools are now offering well organized postgraduate courses. Still fewer offer graduate courses of a quality acceptable to the graduate schools of the universities of which they are a part.

3. A number of deans suggest that when the undergraduate curriculum is decelerated, greater attention can be devoted to refresher courses. They protest the suggestion that the physical facilities and faculties of their schools be burdened with special courses beyond the undergraduate curriculum, and are vigorous in opposition to any arrangement that would schedule such courses "after hours" in relation to the regular schedule.

The questionnaire recently sent out by the War Service Committee of the A.D.A. included a section designed to determine the demand among dental officers for refresher, graduate and postgraduate postwar courses and the particular subjects of special interest to them. The results are interesting. The vast majority asked for postgraduate courses leading to specialization. Only a few indicated the schools in which they preferred to pursue advanced study. While all special areas of dental practice are mentioned in the requests for instruction, the report shows that oral surgery is the most popular subject, followed closely by prosthetics, crown and bridge, orthodontics and anesthesia. Smaller numbers request courses in periodontia, operative dentistry, children's dentistry, x-ray, plastic surgery and public health dentistry.

If the replies of dental officers to this questionnaire can be accepted as true indicators of the demands for advanced study that will be made finally, it appears that the dental schools of the United States cannot hope to meet the situation. To equip dental schools for such work would require a great expansion of physical facilities, equipment and teaching personnel.

COMMITTEE ON LEGISLATION

TRIAL OF MAIL ORDER DENTURE CASES LIMITED TO STATE IN WHICH MAILED

THE United States Supreme Court, in a recent decision, held that, under the provisions of the Traynor Law, a mail order dental laboratory must be brought to trial in the state in which the denture is mailed.

The substance of the majority decision is contained in the following paragraph.

Must these appellees be tried in the Northern district of Illinois or may they be tried in the district of any state through which the dentures were carried including Delaware, the place of delivery? Has Congress authorized such discretion in the enforcement of this act? If it has, there is an end to the matter, for Congress may constitutionally make the practices which led to the Federal Denture Act triable in any federal district through which an offending denture is transported. (Armour Packing Co. *v.* United States, 209 U. S. 56.) An accused is so triable, if a fair reading of the act requires it; but if the enactment reasonably permits the trial of the sender of outlawed dentures to be confined to the district of sending, and that of the importer to the district into which they are brought, such construction should be placed upon the act. Such construction, while not required by the compulsions of Article III, Sec. 2, of the Constitution and of the Sixth Amendment, is more consonant with the considerations of historic experience and policy which underlie those safeguards in the Constitution regarding the trial of crimes.

In the foregoing paragraph, the court asks whether Congress has authorized holding the trial in the state of delivery; admits that, under the Constitution, Congress could do so, but decides that it is more consonant with considerations of historic experience and policy to limit the place of trial to the state in which the dentures are deposited in the mails.

In speaking further on this point, the majority decision also says in part: "It is significant that when Congress desires to give a choice of trial, it does so by specific venue provisions giving jurisdiction to prosecute in any criminal court of the United States through which a process of wrongdoing moves. . . . In the Federal Denture Act, Congress did not make provision for trial in any district through which the goods were shipped."

The majority decision holds that the crime is complete when the dentures are placed in the mails, and therefore a mail order dental laboratory can be prosecuted only in the state in which the dentures are placed in the mails.

In a separate decision is which he concurs with the majority, Justice Murphy states in part:

The Act prohibits the use of the mails for the purpose of sending or bringing into any state certain prohibited articles. It is undisputed that when a defendant places a prohibited article in the mails in Illinois for the purpose of sending it into Delaware, he has completed a statutory offense. Hence he is triable in Illinois; but to hold that the statutory crime also encompasses the receipt of the prohibited article in Delaware, justifying a trial at that point, requires an implication that I am unwilling to make in the absence of more explicit Congressional language.

Very often the difference between liberty and imprisonment in cases where the direct evidence offered by the government and the defendant is evenly balanced depends upon the presence of character witnesses. The defendant is more likely to obtain their presence in the district of his residence, which in this instance is usually the place where the prohibited article is mailed. The inconvenience, expense and loss of time involved in transplanting these witnesses to testify in trials far removed from their homes are often too great to warrant their use. Moreover, they are likely to lose much of their effectiveness before a distant jury that knows nothing of their reputations. Such

factors make it difficult for me to conclude, where Congress has not said so specifically, that we should construe the Federal Denture Act as covering more than the first sufficient and punishable use of the mails insofar as the sender of a prohibited article is concerned. The principle of narrow construction of criminal statutes does not warrant interpreting the "use" of the mails to cover all possible uses in light of the foregoing considerations.

The minority decision written by Mr. Justice Reed follows the argument presented to the Court by the government attorneys and takes an opposite view. The minority states in part:

The statute under consideration condemns the "use" of "the mails or any instrumentality of interstate commerce for the purpose of sending or bringing into any state or territory" any denture which has been made without compliance with the laws of that state or territory, relating to the furnishing of such appliances. The Court narrowly interprets the term "use" to condemn as criminal only the first use of the mails; in this way the Court restricts venue for prosecution to Illinois for trial of any offender who mails a denture in Illinois which is subsequently delivered through "use" of the mails in Delaware. We think, however, that the statute condemns and makes criminal any use of the mails for the prohibited purpose. Under this interpretation the respondent's use of the mails is punishable in Delaware and the dismissal of the information in this case should be reversed.

The venue of a crime may be fixed at any place where the acts denounced as crimes occur. There is no disagreement as to this rule of law. The Court reaches its conclusion upon venue under the Federal Denture Act, not upon any compulsion of Constitution or statute, but because a restriction of the venue to the place of mailing seemed to it more consonant with the underlying purposes of the Constitutional provisions as to venue. These purposes are thought, as the Court expresses it, to include a trial in an environment which is not alien to the accused.

We think the Court misapprehends the purpose of the Constitutional provisions. We understand them to assure a trial in the place where the crime is committed and not to be concerned with the domicile of the criminal nor with his familiarity with the environment of the place of trial. (Haas *v.* Henkel, 216 U.S. 462.) Indeed in the present information nothing appears as to residence or domicile of the accused or as to their place of business.

The crime consists of the use of the mails to send a prohibited denture "into" or bring it "into" another state. The language leads us to the conclusion that a use for the prohibited purpose occurs at whatever place the prescribed denture is handled by the mails or an instrumentality of commerce.

The title of the act indicates that it is directed at practices thought to lead to dental disorders and "to prevent the circumvention of certain state or territorial laws regulating the practice of dentistry." (56 Stat. 1087.) These state laws regulated the fabrication of prosthetic dental appliances. From the hearings it is clear that the purpose of Congress was to protect the public against the evils of ill-fitting dental appliances by restricting interstate commerce to dental appliances which were approved by licensed practitioners of the state into which the appliances were brought. Such was declared to be its purpose by the report of the Senate Committee. (S. Rep. No. 1779, 77th Cong., 2d Sess., p. 1) As the injury would occur normally at the place of delivery and as the act is designed to protect only those states which have laws regulating the furnishing of appliances by unlicensed practitioners, Congress would naturally enact legislation which might punish violations in the state of delivery. The prosecuting officers of that state would be most interested in enforcement and would best understand the scope of the laws of the state of delivery. Congress would not wish to leave immune shipments from foreign countries. (Cf. United States *v.* Freeman, 239 U.S. 117.)

The majority opinion is clearly based on a philosophy of the law rather than on the wording of the Constitution or the Federal Denture Act. In fact, the majority specifically say that their construction of the law is not required by the Constitution, but they feel that their

construction is more in keeping with the underlying spirit of the constitutional provisions concerning the places where trials may be held.

Since the decision in the Delaware case is concerned entirely with the question of where the trial may be held, it will have no effect upon the other case which was brought against the United States Dental Company in Illinois.

BUREAU OF PUBLIC RELATIONS

DENTAL HEALTH EDUCATION IN IOWA

O. E. HOFFMAN,* D.D.S., M.P.H., Des Moines, Iowa

FOR years, the Dental Health Committee of Iowa State Dental Society has sponsored an exhibit at the Iowa State Teachers' Meeting. This year was no exception. The purpose of the exhibit was threefold; namely, to focus the attention of educators on dental health education, to arouse and maintain an interest in an active program and finally to familiarize them with the new educational materials. The Bureau of Dental Hygiene assisted with the plans for the exhibit and with the placing of it in the booth at the Shrine Auditorium in Des Moines. Officials of the Iowa State Department of Health were present at all times to give information and to answer questions during the three-day meeting. The theme of the exhibit was an appeal to educators to build dental health now. The means suggested for motivation were these: (1) active participation in the Iowa Dental Plan; (2) the use of educational aids, and (3) the measuring of results.

In the center panel emphasis was placed on participation in the Iowa Dental Plan, which emphasizes adequate nutrition, oral hygiene and regular periodic visits to the dentist. The Bureau of Dental Hygiene, which is sponsored jointly by the Iowa State Department of Health and the State University of Iowa, has promoted the foregoing program on a state-wide basis since 1927 in its town, parochial and rural schools. While participation is not 100 per cent, the program reached its prewar peak in 1941-1942, when the rural schools in ninety-one counties and 491 town and parochial schools were active. The criteria of "activity" consists in the use of the dental card and the classroom record by the individual school. Both of the above-mentioned materials are shown in the exhibit. A "Handbook of Dental Health Education for the Elementary Teacher," developed by the Bureau of Dental Hygiene personnel, has attained wide distribution. At the present time, 11,000 copies of the publication are in the hands of Iowa teachers and educators.

The panel to the left featured the most recent dental health literature and publications available from the American Dental Association, the National Dental Hygiene Association and the National Dairy Council.

To keep a program alive and functioning efficiently, results must be measured. While it is difficult to measure the results of any health program, it is even more difficult in a dental health educational program. Among the indices used are an accurate record of the number of children who had all necessary dental

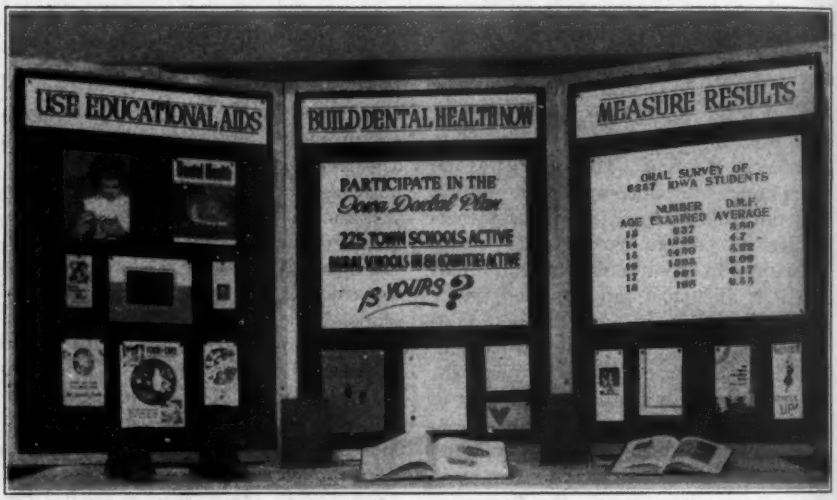
*Secretary, Dental Health Committee; director, Division of Dental Health, Iowa State Department of Health.

cards, and periodic examinations to learn the reduction in the number of lost permanent teeth per hundred children. Both of the methods have been used here. The range in return of dental cards is from 11 to 100 per cent.

The poster displayed in the third panel shows the findings of oral surveys of 6,257 high school students during the past two years. While only the decayed, missing and filled permanent teeth average is shown, the average for each category is available in the different age groups.

The table display consisted of additional educational material. The library of twenty-five slides, most of them in color, is accessible to any individual or organization in the state free of charge, the only cost being that of transportation. The projector for 2 by 2 inch size slides can be obtained at a 50 cent service charge. The books displayed in the exhibit are: "Atlas of the Mouth," by Massler and Schour; "Dentistry for Children," by Brauer, Higley and Boyd; "Feeding the Family," by Mary Swartz Rose, and "Essentials of Nutrition," by Henry Clapp Sherman.

Members of the dental profession in Iowa have given widespread approval and have supported this program through nearly two decades of dental health education. The national recognition which has come to the Iowa Dental Plan is due to the sincere public health interest of those early leaders who had the foresight to plan the program and who set it upon the firm foundation of education, prevention and correction.



Iowa Dental Health Educational Exhibit.

NEWS

BILL TO AID DENTAL RESEARCH AND TO ESTABLISH A NATIONAL INSTITUTE OF DENTAL RESEARCH INTRODUCED BY SEN. JAMES E. MURRAY, JANUARY 10

A BILL authorizing the appropriation of one million dollars for the erection and equipment of a national institute of dental research and $730,000 annually for dental research was introduced January 10 by Sen. James E. Murray, Montana. The research would be conducted under the direction of the Surgeon General of the U. S. Public Health Service at the National Institute for Dental Research, which would be a part of the National Institute of Health, Bethesda, Md., and at certain other private and public agencies.

The text of the bill follows:

A BILL

To provide for, foster, and aid in coordinating research relating to dental diseases and conditions; to establish the National Institute of Dental Research; and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled that, for the purposes of conducting researches, investigations, experiments and studies relating to the cause, diagnosis and treatment of dental diseases and conditions; assisting and fostering similar research activities by other agencies, public and private, and promoting the coordination of all such researches and activities and the useful application of their results, with a view to the development and prompt widespread use of the most effective methods of prevention, diagnosis and treatment of dental diseases and conditions, there is hereby established in the National Institute of Health of the U. S. Public Health Service a division which shall be known as the National Institute of Dental Research (hereinafter referred to as the "Institute").

Sec. 2. The Surgeon General of the Public Health Service (hereinafter referred to as the "Surgeon General") is authorized and directed for the purposes of this Act and subject to its provisions, through the Institute and in cooperation with the National Dental Research Advisory Council hereinafter established

(a) To conduct, assist, and foster researches, investigations, experiments and studies relating to the cause, prevention and methods of diagnosis and treatment of dental diseases and conditions;

(b) To promote the coordination of researches conducted by the Institute and similar researches conducted by other agencies, organizations and individuals;

(c) To provide training and instruction in technical matters relating to the diagnosis and treatment of dental diseases and conditions;

(d) To provide fellowships in the Institute from funds appropriated or donated for such purposes;

(e) To secure for the Institute consultation services and advice of persons who are experts in the field of dental diseases and conditions from the United States and abroad, and

(f) To cooperate with state health agencies in the prevention and control of dental diseases and conditions.

Sec. 3. There is hereby created the National Dental Research Advisory Council (herein referred to as the "Council"), to consist of six members to be appointed by the Surgeon General with the approval of the Federal Security Administrator, and of the Surgeon General, ex officio, who shall be chairman of the Council. The six appointed members shall be selected from leading dental, medical and scientific authorities who are outstanding in the study, diagnosis or treatment of dental diseases and conditions in the United States, and at least four of these members shall be den-

tists who are members of the American Dental Association. Each appointed member shall hold office for a terms of three years, except that (1) any member appointed to fill a vacancy occurring prior to the expiration of the term for which his predecessor was appointed shall be appointed for the remainder of such term and (2) the terms of office of the members first taking office shall expire, as designated by the Surgeon General at the time of appointment, two at the end of the first year, two at the end of the second year and two at the end of the third year after the date of the first meeting of the Council. No appointed member shall be eligible to serve continuously for more than three years, but shall be eligible for a reappointment if he has not served as a member of the Council at any time within twelve months immediately preceding his reappointment. Each appointed member shall receive compensation at the rate of $25 per day during the time spent in attending meetings of the Council and for the time devoted to official business of the Council under this Act, and actual and necessary traveling and subsistence expenses while away from his place of residence upon official business under this Act.

Sec. 4. The Council is authorized

(a) To review research projects or programs submitted to or initiated by it relating to the study of the cause, prevention or methods of diagnosis and treatment of dental diseases and conditions, and certify approval to the Surgeon General for prosecution under section 2 (a) hereof any such projects which it believes show promise of making valuable contributions to human knowledge with respect to the cause, prevention or methods of diagnosis and treatment of dental diseases and conditions;

(b) To collect information as to studies which are being carried on in the United States or any other country as to the cause, prevention and methods of diagnosis and treatment of dental diseases and conditions, by correspondence or by personal investigation of such studies, and with the approval of the Surgeon General make available such information through the appropriate publications for the benefit of health agencies and organizations (public or private), physicians or any other scientists, and for the information of the general public;

(c) To review applications from any university, hospital, laboratory or other institution, whether public or private, or from individuals, for grants-in-aid for research projects relating to dental diseases and conditions, and certify to the Surgeon General its approval of grands-in-aid in the cases of such projects which show promise of making valuable contributions to human knowledge with respect to the cause, prevention or methods of diagnosis or treatment of dental diseases and conditions;

(d) To recommend to the Federal Security Administrator (hereafter referred to as the "Administrator") for acceptance conditional gifts pursuant to section 6; and

(e) To make recommendations to the Surgeon General with respect to carrying out the provisions of this Act.

Sec. 5. In carrying out the provisions of section 2 the Surgeon General is authorized

(a) To provide the necessary facilities where training and instruction may be given in all technical matters relating to diagnosis and treatment of dental diseases and conditions to such persons as in the opinion of the Surgeon General have proper technical training and shall be designated by him for such training or instruction; such persons while receiving training or instruction may, with the approval of the Surgeon General, receive a per diem allowance to be fixed by the Surgeon General, but not to exceed $10;

(b) To establish and maintain, with the approval of the Administrator, research fellowships in the Institute with such stipends or allowances (including traveling and subsistence expenses) as the Surgeon General may deem necessary to procure the assistance of the most brilliant and promising research fellows from the United States or abroad;

(c) To secure for the Institute, from time to time and for such periods as may be advisable, the assistance and advice of experts, scholars and consultants from the United States or abroad who are learned and experienced in the problems involved in accomplishing the purposes of this Act;

(d) To make grants-in-aid for research projects certified by the Council pursuant to section 4 (c), and

(e) To adopt, upon recommendation of

the Council and with the approval of the Administrator, such additional means as the Surgeon General may deem necessary or appropriate to carry out the provisions of sections 1 and 2 of this Act.

Sec. 6. The Administrator is authorized to accept on behalf of the United States gifts made unconditionally by will or otherwise for study, investigation or research into the cause, prevention and methods of diagnosis and treatment of dental diseases and conditions, or for the acquisition of grounds or for the erection, equipment and maintenance of premises, buildings and equipment for the Institute. Conditional gifts may be accepted by the Administrator if recommended by the Surgeon General and the Council. Any such gifts, if in money, shall be held in trusts and shall be invested by the Administrator in securities of the United States, and the principal or income thereof shall be expended by the Surgeon General, with the approval of the Administrator, for the purposes prescribed by this Act, subject to the same examination and audit as provided for appropriations made for the Public Health Service by Congress. Donations of $500,000 or over in aid of research under this Act shall be acknowledged permanently by the establishment within the Institute of suitable memorials to the donors.

Sec. 7 (a) There is hereby authorized to be appropriated a sum not to exceed $1,000,000 for the erection and equipment of a suitable and adequate building and facilities for the use of the Institute in carrying out the provisions of this Act. The Administrator is authorized to acquire, by purchase, condemnation, donation or otherwise, a suitable and adequate site or sites in or near the District of Columbia for such building and facilities, and to erect thereon, furnish and equip such buildings and facilities when funds are made available. No permanent building shall be erected with funds authorized to be appropriated under this Section until after the end of the war.

(b) There is hereby authorized to be appropriated the sum of $730,000 for each fiscal year, beginning with the fiscal year ending June 30, 1945, for the purpose of carrying out the provisions of this Act (except subsection (a) hereof). Sums appropriated pursuant to this subsection may be expended in the District of Columbia for personal services, stenographic recording and translating services, by contract if deemed necessary, without regard to section 3709 of the Revised Statutes; traveling expenses (including the expenses of attendance at meetings when specifically authorized by the Surgeon General); rental, supplies and equipment, purchase and exchange of medical books, books of reference, directories, periodicals, newspapers and press clippings; purchase, operation and maintenance of motor-propelled passenger-carrying vehicles; printing and binding (in addition to that otherwise provided by law), and for all other necessary expenses in carrying out the provisions of this Act.

Sec. 8 (a) There is hereby authorized to be appointed in the Public Health Service, in accordance with applicable law, such commissioned officers as may be necessary to aid in carrying out the provisions of this Act.

(b) This Act shall not be construed as superseding or limiting (1) the functions, under any other Act, of the Public Health Service or any other agency of the United States relating to the study of the prevention, diagnosis and treatment of dental diseases and conditions; or (2) the expenditure of money therefor.

(c) The Surgeon General with the approval of the administrator is authorized to make such rules and regulations as may be necessary to carry out the provisions of this Act.

(d) The Surgeon General shall include in his annual report for transmission to Congress a full report of the administration of this Act, including a detailed statement of receipts and disbursements.

(e) This Act shall take effect thirty days after the date of its enactment.

(f) The term "dental diseases and conditions" shall mean diseases and conditions affecting teeth and their supporting structures and other related diseases of the mouth.

(g) This Act may be cited as the "National Institute of Dental Research Act."

The statement of Senator Murray on the introduction of the bill follows:

I have today introduced in the Senate of the United States a bill to provide for a dental research institute in the United States Public Health Service.

Dental diseases and conditions are the

most common ailments of man. They cause untold suffering and harm. Selective Service examinations showed that defects of the teeth and other dental diseases and conditions were a most common cause of rejection even among the younger men.

In spite of the tremendous advances of science, the causes of dental decay are partly or largely unknown. The methods of prevention, diagnosis and treatment for many dental conditions are still limited or uncertain in value.

Modern dentistry is just about one hundred years old. In the course of that century, dentistry has made tremendous progress. American dentistry is second to none in the world. Still our dental profession and our public health leaders are not satisfied. They are anxious to speed up the progress of scientific knowledge, so that dentists and other health professions will be able to do vastly more than they can do today in preventing dental diseases and curing what cannot be prevented. One of the most important and promising ways to progress is through well-equipped research.

The dental profession of the United States strongly supports the proposals of this bill. I am sure that many other professional groups that are interested in the improvement of health will also support it as a much needed and useful step. To the public, this dental research program offers the hope of bringing, in the future, immeasurable relief from suffering, and tremendous improvement in positive health, in return for expenditures that would be relatively trivial.

My bill proposes that there should be established a national Institute of Dental Research, as a division of the National Institute of Health in the United States Public Health Service. The dental research that is already being done by the Public Health Service and other agencies, public or private, would be greatly increased under arrangements that assure coordination of the new and the old activities.

The Surgeon General of the Public Health Service is to direct the new institute and its work. To assist him, there would be created a national Advisory Dental Research Council, to include—in addition to the Surgeon General, who will be chairman—six appointed members—at least four of whom shall be dentists—to be selected from leading authorities.

The institute is to be conduct and aid research on the cause, prevention and methods of diagnosis and treatment of dental diseases and conditions; to promote coordination of dental research; to provide fellowships in the institute and make grants-in-aid to universities and other institutions; to secure the consultation services of other experts, and to cooperate with state health agencies.

The institute is authorized to receive gifts from private sources.

The Surgeon General is required to make a full report to Congress each year.

To give effect to this program, the bill proposes that Congress should authorize appropriations, up to a maximum of $1,000,000 to erect and equip a suitable building after the war; and up to maximum of $730,000 a year to provide a budget to carry out the program.

Dental diseases and conditions, in damage to health and working power, now cost untold amounts every year, amounts which are probably to be measured in billions of dollars, to say nothing of human suffering and unhappiness. In addition, the people of the United States spend several hundred million dollars a year on dental services, even though they are receiving only a minor part of the dental care that they really need. It is, therefore, good economy and good sense that we should invest an amount equal to a very small fraction of these sums in learning more about the causes of dental diseases, in inventing new and better methods of treatment and in improving the efficiency and economy of dental care.

This is a bill proposing that we should make a good investment in the future health of the people. It should be promptly enacted by Congress.

FIFTH ANNUAL CHILDREN'S DENTAL HEALTH DAY SPONSORED BY THE CLEVELAND DENTAL SOCIETY

THE Cleveland Dental Society will present its Fifth Annual Children's Dental Health Day at the Hotel Carter, Cleveland, February 5. A program of essays and clinics on preventive and children's dentistry has been arranged for the all-day session, which will end with the dinner meeting. The meeting will be under the general direction of C. F. Tuma and H. C. Janke, with E. Carl Miller chairman of the program committee. The program of the meeting follows:

Morning Session: 8:45 a.m., Address of Welcome, James V. Gentilly, president, Cleveland Dental Society; 9:00 a.m., "The Functional Devitalized Tooth," Alfred E. Seyler, Detroit, Mich.; 10:00 a.m., "Prevention—the Answer to Dentistry's Problems," Walter H. Scherer, Houston, Texas, President, A.D.A.; 11:00 a.m., "Operative Technics for Tooth and Child," Ralph L. Ireland, Lincoln, Nebr.

Midday Session: 12:15 p.m., luncheon, Ralph E. Creig, toastmaster; presentation of distinguished guests; 1:15 p.m., "Caries Can Be Controlled," F. A. Arnold, Jr., Bethesda, Md.; 1:45 p.m., A Cappella Choir.

Afternoon Session: 2:00 to 5:00 p.m., limited attendance clinics: "Pulpotomies of Primary Teeth," A. E. Seyler; "Dentistry for the Child," Ralph L. Ireland; "Practical Preventive Measures," F. A. Arnold; "Orthodontic Problems," Leuman M. Waugh; "Correction of Speech Defects," H. K. Cooper; "Local Anesthesia for the Child," J. N. Gasaway; 2:00 to 4:00 p.m., postgraduate clinics; 4:45 to 5:30 p.m., "Dental Correction of Speech Defects," H. K. Cooper, Lancaster, Pa.

Evening Session: 6:00 p.m., dinner, C. F. Tuma, chairman; 7:00 p.m., "Practical Application of Prevention in Orthodontics," Leuman M. Waugh, New York. There will be an all-day exhibit of educational material.

THIRD WAR CONFERENCE

The Third War Conference on Industrial Health will be held April 23-28 at the Drake Hotel, Chicago. This is a joint meeting of the American Association of Industrial Physicians and Surgeons, the American Industrial Hygiene Association, the National Conference of Governmental Industrial Hygienists, the American Association of Industrial Nurses and the American Association of Industrial Dentists. The meeting will feature essays and clinics dealing with all phases of industrial health as well as comprehensive scientific and commercial exhibits. The industrial dentists will devote Wednesday, April 25, to a full-day scientific program. All American Dental Association members interested in industrial health are invited. Ernest Goldhorn, Chicago, is president of the association.

DEATHS

ALLEN, RUSSELL F., Monticello, N. Y.; Pennsylvania College of Dental Surgery, 1890; died December 6; aged 76.

APP, W. H., Decatur, Ill.; Chicago College of Dental Surgery, Dental Department of Loyola University, 1901; died November 19; aged 67.

ARDNER, ANDREW J., Chicago, Ill.; Chicago College of Dental Surgery, Dental Department of Loyola University, 1900; died November 12.

ARMSTRONG, WILLIAM S., San Diego, Calif.; Ohio College of Dental Surgery, Dental Department of the University of Cincinnati, 1911; died November 24.

AUSTIN, J. L., Chattanooga, Tenn.; Vanderbilt University, School of Dentistry, 1906; died October 9; aged 65.

BARONE, ANTHONY, Boston, Mass.; Tufts College Dental School, 1917; died December 8; aged 48.

BERRY, CHARLES W., Portland, Maine; Tufts College Dental School, 1915; died December 20; aged 66.

BLACHLEY, CARL WENTWORTH, Centerville, Iowa; Des Moines College of Dental Surgery, 1902; died October 24; aged 66.

BLAKER, CHARLES S., Philadelphia, Pa.; Thomas W. Evans Museum and Dental Institute School of Dentistry, 1896; died November 22; aged 70.

BOYLHART, FREDERICK, Brooklyn, N. Y.; New York School of Dentistry, 1898; died December 5; aged 72.

BRICKELMAIER, GEORGE, Haverstraw, N. Y.; New York College of Dentistry, 1913; died November 18; aged 52.

BROCKETT, HOLLY, Kansas City, Mo.; Western Dental College, Kansas City, 1906; died November 13; aged 64.

BROWN, E. NORMAN, Los Angeles, Calif.; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania, 1917; died November 11; aged 54.

BRUCE, G. R., Pasadena, Calif.; State University of Iowa, College of Dentistry, 1899; died November 21.

CARTWRIGHT, CHARLES HULL, Hollywood, Calif.; Northwestern University Dental School, 1910; died September 25; aged 65.

CLARK, WILLIAM H., Manitowoc, Wis.; Chicago College of Dental Surgery, Dental Department of Loyola University, 1903; died November 16; aged 65.

DALE, WILLIAM E., Altoona, Pa.; Temple University School of Dentistry, 1905; died November 30.

DE LAP, R. H., Kelso, Wash.; Department of Dental Surgery, Wisconsin College of Physicians and Surgeons; died November 15; aged 67.

DELCHER, JOHN E., Toledo, Ohio; Western Reserve University Dental School, 1935; died December 10; aged 36.

DEVANEY, WILLIAM L., Robbinsdale, Minn.; State University of Iowa, College of Dentistry, 1900; died December 14; aged 70.

DURGIN, OLIVER K., Winnebago, Minn.; Tufts College Dental School, 1903; died November 25; aged 64.

EDGE, CHARLES H., Houston, Texas; Texas Dental College, Houston, 1907; died November 13; aged 70.

EDSALL, ALVAH W., Warwick, N. Y.; Temple University School of Dentistry, 1883; died December 11; aged 83.

EGGEMAN, GODFREY, Denver, Colo.; Indiana University School of Dentistry, 1890; died November 3; aged 69.

EMERSON, CHARLES J., Omaha, Nebr.; Creighton University, College of Dentistry, 1910; died November 9; aged 61.

EVERHART, DAVID G., Frederick, Md.; University of Maryland, School of Dentistry and the Baltimore College of Dental Surgery, 1910; died December 3; aged 54.

FONNER, LYNN A., Fort Wayne, Ind.; Indiana University School of Dentistry, 1914; died December 7; aged 51.

FREER, MAX E., Pico, Calif.; University of Southern California, College of Dentistry, 1926; died November 14; aged 42.

GA NUN, C. FRED, New York, N. Y.; New York University, College of Dentistry, 1928; died December 2; aged 40.

GIDDES, ELMER A., Plainfield, N. J.; New

York College of Dentistry, 1900; died December 4; aged 66.

GIER, S. WILLIAM, Los Angeles, Calif.; died December 16; aged 83.

GOODWIN, CAROLYN M., Waukesha, Wis.; died November 19; aged 72.

GRANT, L. F., Munfordsville, Ky.; University of Louisville, School of Dentistry, 1905; died November 23; aged 65.

GRIMMER, ANGELO HENRY, Covington, La.; Tulane University of Louisiana, College of Medicine, School of Dentistry, 1905; died November 30; aged 78.

HALLETT, WILLIAM S., Wilmington, Del.; Pennsylvania College of Dental Surgery, 1897; died December 10; aged 69.

HARDON, CHARLES M., Mansfield, Mass.; Tufts College Dental School, 1899; died November 11; aged 67.

HARMON, JAMES B., Chicago, Ill.; Chicago College of Dental Surgery, Dental Department of Loyola University, 1904; died December 5.

HARPER, CHARLES ERNEST, Danville, Va.; University of Maryland, School of Dentistry and the Baltimore College of Dental Surgery, 1883; died December 12; aged 80.

HARRIS, ABRAHAM H., Evanston, Ill.; Northwestern University Dental School, 1919; died November 19.

HENDRICK, JAMES G., Los Angeles, Calif.; died November 11; aged 70.

HOURN, GEORGE E., St. Louis, Mo.; University of Minnesota, College of Dentistry; died December 10; aged 66.

HOYE, FREDERIC J., New Bedford, Mass.; Tufts College Dental School; died December 10; aged 58.

HUGHEY, G. M., Buffalo, N. Y.; University of Buffalo, School of Dentistry, 1907; died November 11; aged 59.

HYNES, WILLIAM, Elizabethtown, Ky.; University of Louisville, School of Dentistry, 1897; died November 15; aged 68.

JACKSON, J. HOLMES, Burlington, Vt.; Temple University School of Dentistry, 1890; died December 15; aged 73.

JOHNSON, FRANK M., Evanston, Ill.; Chicago College of Dental Surgery, Dental Department of Loyola University, 1899; died December 14; aged 81.

JOHNSTON, ELOF T., Jamestown, N. Y.; Detroit College of Medicine, Department of Dental Surgery, 1907; died December 4; aged 67.

KALINOWSKI, STANLEY L., Pittsburgh, Pa.; University of Pittsburgh, School of Dentistry, 1918; died November 26; aged 50.

LANIGAN, FRANCIS J., Cambridge, Mass.; Tufts College Dental School, 1903; died November 20; aged 71.

LIEF, NATHANIEL, New York, N. Y.; New York College of Dentistry, 1914; died December 22; aged 51.

LUCKIE, S. BLAIR, Chester, Pa.; Philadelphia Dental College, 1882; died December 21; aged 96.

MCCLOUGHAN, GEORGE H., Newton, N. J.; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania, 1900; died November 23; aged 65.

MCCLURE, CHARLES J., New York, N. Y.; University of Buffalo, School of Dentistry, 1897; died December 1.

MCKINLEY, WILLIAM J., Philadelphia, Pa.; Medico-Chirurgical College of Philadelphia, 1903; died November 16; aged 62.

MCLEAN, HENRY M., Spartanburg, N. C.; University of Maryland, School of Dentistry and the Baltimore College of Dental Surgery, 1915; died December 4; aged 54.

MANN, GEORGE H., St. Johns, Mich.; University of Michigan, College of Dental Surgery, 1893; died November 21; aged 71.

MANNING, J. HENRY, Providence, R. I.; Tufts College Dental School, 1899; died November 22.

MATHER, WILLIAM J., Des Moines, Iowa; Western Dental College, Kansas City, 1906; died October 1; aged 70.

MILLER, FRANK H., Highland Park, Mich.; died November 1; aged 65.

MOITH, FERDINAND, Beacon, N. Y.; New York College of Dentistry, 1887; died December 2; aged 84.

MORRIS, CHESTER A., Tombstone, Ariz.; North Pacific College of Oregon, School of Dentistry, 1905; died December 19; aged 67.

MYERS, JOHN G., Richmond Hill, N. Y.; died December 2; aged 53.

NELSON, PERRY H., Lake Worth, Fla.; Marquette University, School of Dentistry, 1912; died November 27; aged 58.

O'TOOLE, CLEMENT B., Staten Island, N. Y.; School of Dentistry, Georgetown Univer-

sity, 1937; died December 15; aged 34.

PELOT, THOMAS C., Atlanta, Ga.; Atlanta Dental College, 1908; died December 12; aged 73.

PERLEE, GEORGE W., Indianapolis, Ind.; Indiana University School of Dentistry, 1910; died December 17.

PETERSON, PAUL H., St. Paul, Minn.; University of Minnesota, College of Dentistry, 1922; died December 23; aged 45.

PRINTY, GEORGE E., Creighton, Nebr., Creighton University, College of Dentistry, 1915; died September 27.

REYHER, MAX, Belmar, N. J.; died December 6; aged 82.

ROTNER, PHILIP, Yonkers, N. Y.; College of Dental and Oral Surgery, 1922; died December 5; aged 45.

SCHOLL, FRED C., Indian Head, Md.; died December 15; aged 67.

SCHUSLER, PHILIP J., Franklin, Pa.; died December 15; aged 72.

SCHWERING, LESLIE, Eugene, Ore.; North Pacific College of Oregon, School of Dentistry, 1923; died November 14.

SCOTT, JAMES O., Grand Rapids, Mich.; Detroit College of Medicine, Department of Dental Surgery, 1901; died December 5; aged 73.

SHINNESS, B. S., Seymour, Ind.; University of Louisville, School of Dentistry, 1896; died December 6; aged 70.

SMITH, CHARLES D., Angels Camp, Calif.; died October 27; aged 86.

SPEAR, EDWARD C., Newcomerstown, Ohio; Ohio State University, College of Dentistry, 1909; died November 21; aged 63.

STACY, JAMES H., Lexington, Ky.; University of Louisville, School of Dentistry, 1931; died November 17; aged 38.

STANLEY, CHARLES C., Columbia, S. C.; University of Maryland, School of Dentistry and the Baltimore College of Dental Surgery, 1894; died November 9; aged 74.

STARBUCK, WILLIAM R., Bricelyn, Minn.; State University of Iowa, College of Dentistry, 1904; died October 19; aged 63.

THOMAS, J. A., Albert Lea, Minn.; University of Minnesota, College of Dentistry, 1911; died December 31.

VEDDER, F. F., Los Angeles, Calif.; died November 17.

WHERRY, ARTHUR C., Salt Lake City, Utah; Chicago College of Dental Surgery, 1902; died December 26; aged 64.

WINBURN, WALTER J., Winchester, Ky.; University of Louisville, School of Dentistry, 1906; died November 15; aged 51.

WOOD, HARRY E., Miami, Fla.; Atlanta-Southern Dental College; died November 26 at Ft. Lewis, Wash.; aged 49; Dr. Wood was a a captain in the U. S. Army Dental Corps.

YAMAMOTO, TATSUO, Des Moines, Iowa, College of Physicians and Surgeons of San Francisco, a School of Dentistry, 1906; died December 4; aged 67.

YOUNG, GERHART H., Evanston, Ill.; Northwestern University Dental School, 1903; died September 13; aged 72.

LIST OF CERTIFIED DENTAL MATERIALS

THE list of certified dental materials published in the January 1 issue should have included, under "Certified Denture Resins," Justi-Tone, pink, powder-liquid, H. D. Justi & Son, Inc., following "Densene." An addition to the list, under "Certified Inlay Casting Waxes," to follow "Kerr-Blue—Hard," is Maves, The Ohio Chemical & Mfg. Co.

CURRENT LITERATURE

Journal of Oral Surgery
July 1944

Titles marked with an asterisk are reviewed below.

*The Development of the Treatment of Jaw Fractures. L. Laszlo Schwartz, D.D.S., New York, N. Y.—193.

*Soft Tissue Surgery. B. E. Luck, D.D.S., Lansing, Mich.—222.

*Cleft Palate. John W. Kemper, D.D.S., Ann Arbor, Mich.—227.

*Osteotomy for the Correction of Mandibular Malrelation of Developmental Origin. Reed O. Dingman, A.B., D.D.S., M.S., M.D., Ann Arbor, Mich.—239.

*A Plastic Operation for Lengthening the Congenitally Short Upper Lip: Preliminary Report. John F. Ford, M.D., New York, N. Y.—260.

*Monocytic Leukemia with Oral Manifestations: Report of Case. Lyle F. Aseltine, D.D.S., Ann Arbor, Mich.—266.

*Maxillary Cyst: Report of a Case. Frederic W. Coggan, D.D.S., Kalamazoo, Mich.—268.

*Ludwig's Angina and Anesthetic Complications. Victor H. Frank, D.D.S., Philadelphia, Pa.—271.

*Ameloblastoma: Report of Case. Norval H. McDonald, D.D.S., Richmond, Va.—275.

*Multiple Cementoma. James L. Bradley, D.D.S., M.S.D., Bethesda, Md.—278.

Book Reviews—283.

The Development of the Treatment of Jaw Fractures

L. Laszlo Schwartz

Schwartz writes very interestingly and completely on the evolution of jaw fracture treatment. Wars, throughout history, have played a prominent part in the advancement of care of facial injuries. Progress in this field did not occur as the result of new and basic discoveries, but rather through the improvement of known methods. "The knowledge of occlusion belonging to dental science provided an accurate means of determining the exact extent of deformity following fracture of the jaw. The mechanical ingenuity and dexterity resulting from dental training were the implements for preventing resultant deformity." During the Civil War, great strides were made, and dentistry was brought into the field. "Moreover, the contributions of dentistry to the treatment of jaw fractures, during the Civil War, exerted another influence. These achievements, through the recognition accorded them, undoubtedly played an important part in establishing the relationship of dentistry to the new specialty of oral surgery which flourished with the work of James E. Garretson after the war. This relationship was described by Cryer toward the close of the century when he pointed out that oral surgery 'is perhaps unique in that, while really a department of surgery, the educational approach to it is through dentistry, and not through medicine'."

Soft Tissue Surgery

B. E. Luck

Luck discusses and illustrates soft tissue flaps and incisions as used about the teeth. Any incision should be so located that the resultant flap margins are over neutral bone, preventing collapse of the flap into the wound when it is replaced. Preference is indicated for a reflected gingivomucoperiosteal flap which eliminates, as nearly as possible, vertical types of incisions. "Care is taken to include the periosteum; the entire mass of soft tissue being detached from its gingival attachment around the neck of the molar tooth." In summarizing, it is stated that (1) adequate exposure is the key to success in the removal of fractured roots, (2) conservative measures in dealing with soft tissues and osseous supporting tissues prevent postoperative complications, and (3) gingival mucoperiosteal flap construction eliminates unnecessary incisions, creates less trauma, preserves nerve and blood supply to the tissues and provides adequate exposure for most minor oral surgical procedures.

Osteotomy for the Correction of Mandibular Malrelation of Developmental Origin

REED O. DINGMAN

DINGMAN reports six cases of mandibular prognathia and retrognathia and discusses and describes corrections by osteotomy. Section should be through the body of the bone if the distance to be negotiated is more than two-thirds the width of the ramus or if impingement of the lower fragment against the mastoid process is imminent. If the maxillary and mandibular arches are in proportion, section of the ramus is preferable. Orthodontic appliances fastened to all available teeth, except the incisors, and intermaxillary elastics were used for immobilization, which averaged eight weeks. The Stout method of fixation was employed satisfactorily in one case, but preference is expressed for orthodontic bands. Orthodontic treatment is sometimes indicated prior to operation and no case is treated surgically if orthodontia may solve the problem. Preoperative casts of the teeth are essential to diagnosis and treatment planning. Usually, the ramus is sectioned downward and backward in prognathia and downward and forward in retrognathia, but a study of the case is necessary first to prevent elongation of the ramus or nonunion. Questionable teeth must be treated before operation. Osteotomy of the ramus is performed above the mandibular foramen. Rubber bands are placed interdentally before operation. The open, extra-oral approach back of the angle is preferred. A pathfinder needle is used to carry a Gigli saw, which separates the ramus. After the cut has been made on the first side, holes are drilled through the abutting fragments and 20-gage stainless steel wire is properly placed, but not twisted down until the opposite side has been completed. Both sides are then immobilized in their new positions by the applied wires. Open bite, encountered postoperatively on occasion, can be serious enough to require additional operative measures subsequently.

Cleft Palate

JOHN W. KEMPER

KEMPER considers the complete treatment of cleft palate, not only from the surgical aspect, but also from the psychologic and functional sides. Evaluation of the success of surgical treatment must be based wholly on the functional result. Lacking the coordination of all factors, a surgical triumph may turn out to be a failure. The treatment of cleft palate begins at birth. "The first important step is to attempt to dispel immediately the initial emotional reaction of despair and disappointment of the parents by acquainting them with the actual facts concerning the child's deformity, and to give them a hopeful outlook regarding its correction." The influence of heredity on the incidence of cleft palate is not conclusive, but is important, no doubt. Early feeding is accomplished by any method which deposits the food on the posterior part of the tongue. A medicine dropper or ear bulb may be useful at first. Use of the bottle should be started as soon as possible. If the hole in the nipple is enlarged sufficiently to provide a gravity drip, the baby can take the food satisfactorily. Soft and solid foods should follow at the proper age. Pediatric care is fundamental. Repair of the lip provides sufficient pressure to close the alveolar cleft. The whole cleft palate procedure should be completed before or at the time speech habits are being formed—usually between the fifteenth and the twenty-fourth month. Two or more stages are employed in closing the clefts. The first operation approximates the hard palate area, utilizing sliding flaps anteriorly from the aponeurotic attachment. The second procedure is begun by incisions down the pterygomandibular folds, followed by fracture of the hamular processes, dissection of the aponeurotic and nasal mucosal attachments, paring of the medial surfaces of the soft palate and suturing. Buried sutures are used sparingly. Coaptation is by mattress and interrupted sutures. Short palates are lengthened by preserving the palatine vessels while stretching them and using the double V pushback. Speech training and orthodontic procedures complete the treatment.

A Plastic Operation for Lengthening the Congenitally Short Upper Lip: Preliminary Report

JOHN F. FORD

FORD describes an operation for lengthening the congenitally short upper lip. If

upper teeth and gums are exposed, the case is one suitable for plastic procedure. Lengthening of the short lip has esthetic value as well as affording functional benefits, including reduction of mouth-breathing and prevention of maxillary overgrowth. "Hemostatic lip clamps, which are applied to each side of the upper lip, greatly diminish bleeding during the operation and make identification of the muscles easier. An incision is made within the mouth, along the roof of the superior vestibule and near the maxillary border, extending the distance between the premolar teeth and upward to the anterior spine of the maxilla. *M. depressor septi, nasalis, incisivus superioris* and *nasolabialis* and the three heads of *quadratus labii superioris* are sectioned along a horizontal line with the lower border of the anterior nasal spine of the maxilla, this procedure being carried out on each side. Through this incision, the skin and mucous membrane are undermined downward for a distance of about 1 cm. This undermining seems to be of benefit in preventing postoperative lip retraction and gives a better lip contour." Care must be taken not to incise the zygomatic muscle. This operation produces a nearly normal lip in the case of moderate shortening and improvement in more extreme cases.

Monocytic Leukemia with Oral Manifestations: Report of Case

LYLE F. ASELTINE

ASELTINE reports a case of monocytic leukemia with oral manifestations. Vincent's infection was the first complaint. Eight months before, the patient had had a "cold," followed by anorexia, loss of weight and lassitude. A loose tooth was removed and the extraction site refused to heal. The oral mucous membranes were pale. Blood examination revealed the leukemia. "It is important that the dentist be alert for such signs and symptoms as have just been described; that is, progressive weight loss, persistent oral ulceration, malaise, pallor and skin lesions. Any one or a combination of these should arouse suspicion and lead one to rule out the possibility of a blood dyscrasia."

Maxillary Cyst: Report of a Case

FREDERIC W. COGGAN

COGGAN reports an interesting case of cyst produced by the presence of bismuth paste. Six years before, the patient had a supernumerary tooth removed from the upper incisor area and bismuth paste was closed in the wound. A rather extensive cystic reaction resulted.

Ludwig's Angina and Anesthetic Complications

VICTOR H. FRANK

FRANK reports a case of Ludwig's angina with anesthetic difficulties. Tracheotomy, incision and drainage during collapse without anesthesia were accomplished. The important observation is made that preoperative use of morphine may have been an important factor in the respiratory difficulties encountered during the operative procedures. It is suggested that nasal intubation and placing of the tube in the larynx should be used in preoperative preparation.

Ameloblastoma: Report of Case

NORVAL H. MCDONALD

MCDONALD reports a case of adamantinoma, believed to be a dentigerous cyst about an unerupted lower third molar. The only clinical sign was a "pebbled area" distally from the last visible molar. Postoperative x-ray films taken at six-months intervals were suggested.

Multiple Cementoma

JAMES L. BRADLEY

BRADLEY reports two cases of multiple cementomas. "This tumor is the result of a benign fibroblastic proliferation of the cementoblastic layer of the periodontal membrane in the apical region of the tooth. The soft-tissue fibroblastic activity represents the first stage of the cementoma formation. Initially, there is a resorption of the periapical bone, which is followed, in later stages, by cementum deposition, and eventually by the development of more mature tumors or hypercalcification. Roentgenographically, the first, or fibroblastic, stage resembles a dental granuloma or radicular root cyst. In the second stage, enough cementum has formed to produce a radiopaque area within a larger

radiolucent area. The third stage is more matured, presenting a radiopaque mass surrounded by a narrow, fairly even radiolucent band. Clinically, the teeth are vital and normal in color, and digital examination in some cases reveals small rounded bony masses. Apparently, no cases of metastasis of these neoplasms have been reported." Roentgenographically, it is impossible to differentiate early cementomas from many other diseases producing radiolucency. Later cases show irregular calcification. Most reported cases of cementoma occur in women, as did both of these.

Journal of Dental Research

August 1944

Subjects marked with an asterisk are reviewed below.

*The Pathogenesis of Enamel Hypoplasia: An Experimental Study. Seymour J. Kreshover, Yale University—231.

Ultra Microstructures of the Human Tooth as Revealed by the Electron Microscope. Charles H. Gerould, Midland, Mich.—239.

*Domestic Water and Dental Caries. VI. The Relation of Fluoride Domestic Waters to Permanent Tooth Eruption. Edwin M. Short, National Institute of Health—247.

*Intra-Oral Hydrogen-Ion Concentrations Associated with Dental Caries Activity. R. M. Stephan, University of Chicago—257.

*A Colorimetric Method for the Determination of Amylolysis of Saliva. Ivan Pratt and L. L. Eisenbrandt, University of Kansas City—267.

*The Patterns of Growth and Development of the Human Teeth and Jaws. Moses Diamond, Columbia University—273.

The Pathogenesis of Enamel Hypoplasia: An Experimental Study.

SEYMOUR J. KRESHOVER

THE present study was undertaken to provide an experimental check on conclusions drawn from previously reported findings on human teeth which permitted tracing of the pathogenesis of enamel hypoplasia from its earliest manifestations to the end-result of clinically demonstrable defects. The method of approach was to so choose the type of etiologic agent and susceptible host that the severity of injury to the developing teeth could be controlled and thereby be compared with the condition in human beings.

The low degree of susceptibility of mice to experimental tuberculosis favored the possibility that resulting dental abnormalities would be mild, whereas the guinea-pig, being more susceptible to the disease, would probably show more severe changes. Seventy mice and fourteen guinea-pigs were infected for this purpose. Control animals were employed in each group.

The similarity of the observed dental changes to those previously reported in human material makes possible the experimental confirmation of the proffered concept regarding the sequence of histologic changes involved in the pathogenesis of hypoplastic enamel. These are: abnormal formation or secretion of enamel-like substance subjacent to and within the cell cytoplasm; beginning vacuolization of ameloblasts, complete cystic destruction of ameloblasts and cessation of enamel formation resulting in notchlike defects.

Domestic Water and Dental Caries. VI. The Relation of Fluoride Domestic Waters to Permanent Tooth Eruption.

EDWIN M. SHORT

PERMANENT tooth eruption rates of 4,453 children aged 12 to 14 years in twelve communities in Illinois and Colorado have been determined, with comparisons made on the basis of the fluorine content of the public water supply. The content of water supplies varied between 0.0 and 2.6 parts per million. In contrast to children using fluoride free water, those using water containing 2.6 parts per million showed a significant delay in tooth eruption. No significant difference was noted in children using water below 2.6 parts per million.

Intra-Oral Hydrogen-Ion Concentrations Associated with Dental Caries Activity.

ROBERT M. STEPHAN

IN this study of sixty-five individuals, it was found that there was an increase in the acidity of bacterial material on the teeth in every case after the mouth was rinsed with a glucose solution. However, the extent and duration of this increase in acidity (drop

in pH) varied with the degree of caries activity, being greatest in cases with extensive caries activity. An acidity below pH 5 occurred only in cases with some caries activity, and the occurrence of these low pH values was most frequent in cases of greatest caries activity. The pH drop was more marked on the labial surfaces of the upper teeth than on the labial surfaces of the lower teeth, and this difference corresponded with the relative susceptibility to caries of these tooth surfaces.

A Colorimetric Method for the Determination of Amylolysis of Saliva.

IVAN PRATT and L. L. EISENBRANDT

SINCE methods for estimating the rate of amylolysis in saliva are not quantitatively satisfactory, a procedure was developed using a Klett-Summerson photo-electric colorimeter (KS-54 filter) and a standardized set of conditions. The dial readings were calibrated for equivalent milligrams of starch. Soluble starch was accurately weighed, and then dissolved in water so that 40 ml. contained 3.5 mg. of starch. The indicator was 0.05 normal iodine.

To the starch solution in a 20 mm. cell was added 0.1 ml. of unstimulated saliva, the instrument was adjusted to zero and, after digesting for one minute, 0.025 ml. of iodine solution was added and a dial reading was taken which showed the amount of undigested starch. This amount, subtracted from the original 3.5 mg., gave the quantity of starch digested. The greatest difficulty experienced was in obtaining standard starch solutions. Usually, this factor has been inadequately considered.

In addition to describing an improved technic, the amylolytic activity of unstimulated saliva of six subjects was tested at 9 and 11 a.m. and 1, 3 and 5 p.m. for five successive days. The data, studied statistically, showed that subjects varied in amylolytic activity. When the subjects were considered collectively, it was found that the group did not vary significantly during the day.

The Patterns of Growth and Development of the Human Teeth and of the Jaw Bones.

MOSES DIAMOND

HISTOLOGIC studies were made upon extensive human embryologic and fetal heads. The material was prepared in serial sections and cut in horizontal, sagittal and frontal directions. Bones of the head were examined, including suture relations to determine directions of growth. Growth of the crown germs was also examined. The following conclusions were arrived at: The patterns of growth of the jaw bones are entirely separate from the patterns of growth of the dentures without any dependence of one upon the other. Growth of the jaw bones may continue in all directions whether teeth are present or congenitally absent. The only relation between the patterns of growth is spatial. Sutural growth does not result in enlargement by the spreading of adjacent bones. Enlargement occurs by the usual processes of apposition and resorption, the suture space shifting in space with the growing bones. Additional observations revealed that the dental follicle is an important factor in the development of the crown of the tooth. The dental follicle grows, the surrounding bone resorbing until a crypt is established large enough to accommodate the fully grown crown of a tooth. Epithelium of the crown germ continuously invades the connective tissue of the dental follicle. During this stage, there is no migration of the crown germ as is commonly believed, but it remains instead sheltered in a fixed site until the enamel of the crown is virtually completed.

"Fluorine-Like" Action of Various Substances on the Teeth.

J. T. IRVING

YOUNG rats on a normal diet (calcium: phosphorus 1.46) were given cutaneously injections of sodium fluoride, potassium oxalate, sodium citrate, manganese sulfate, calcium chloride, strontium chloride, disodium hydrogen phosphate, magnesium chloride, sodium molybdate, aluminum chloride, beryllium nitrate, lead nitrate and borax. In all cases, the animal received 100-200 mg. of the element concerned, except fluorine and beryllium, dosage of which was greatly reduced, according to animal tolerance. The amounts of oxalate and citrate injected were equivalent in calcium combining power to the amount of fluorine injected. Rats were sacrificed two days after injection. In no case except that of fluorine was any

effect on the organic enamel found. Injection of oxalate, manganese, calcium, phosphorus, magnesium, molybdenum and beryllium had typical fluorine-like effects in dentin. Citrate, lead and boron injection had no effect. Rats on a diet (calcium:phosphorous 0.27) were transferred to this régime at between 50 and 60 gm. in weight and were maintained on it for twenty-eight days. At this time, they were given injections of all of the substances mentioned except magnesium chloride, and killed four days later. Apart from fluorine, only phosphorus produced a continuous line in predentin. Calcium injection caused a healing process as occurs when the calcium:phosphorus ratio of the diet is adjusted. The first action of fluorine appears to be directly on the enamel organ. A second influence, like that of the other substances examined, may involve alteration of blood composition. —*Nature*, 154:149, 1944.

J. E. GILDA.

Rheumatic Fever in Cincinnati in Relation to Rentals, Crowding, Density of Population and Negroes.

A. G. WEDUM and B. G. WEDUM

THIS survey emphasizes the importance of poverty and crowding in the genesis of rheumatic fever, but these conditions are considered indicative of a more fundamental epidemiologic principle. Comparison of findings is made with what is known about rheumatic infections in military camps. In Colorado, Idaho and Utah, the camps are in high areas, well removed from water. Housing, sanitation and diet are good. Only two features are common to military and civilian population: (1) crowding and (2) inability to control the microclimate. It is often impossible for either the poor or the military man to rest when tired, get warm when cold or keep dry when it is rainy.—*Am. J. Pub. Health*, 34:1065, October 1944.

PAUL H. KEYES.

Journal of Dental Research

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*The Naso-Meatal Line as a Guide for the Determination of the Occlusal Plane. Wendell L. Wylie (University of California)—305.

Sodium Hexametaphosphate as an Aid in the Treatment of Periodontal Disease. Donald A. Kerr and Henry Field, Jr. (University of Michigan)—309.

*Diabetes in Relation to Certain Oral and Systemic Problems. Daniel E. Ziskin, Eli H. Siegel, and Winifred C. Loughlin (Columbia University and New York Diabetes Association)—317.

*Reestablishment of Transseptal Fibers Following Extraction. Samuel W. Chase and John Revesz (Western Reserve University)—333.

*The Pain Threshold Raising Effects of Amphetamine. D. Y. Burrill, F. R. Goetzl, and A. C. Ivy (Northwestern University Medical School)—337.

*Experimental Papillectomy, Part II: Histological Study (Preliminary Report). William Lefkowitz, Charles F. Bodecker, and Harry H. Shapiro (Columbia University)—345.

*Studies on the pH of Saliva. L. L. Eisenbrandt (University of Kansas City)—363.

*Mutations of Bacteria Under Dietary Influence. P. H. Belding and L. J. Belding, Waucoma, Iowa—375.

*Histopathology of Enamel Caries. Bernhard Gottlieb (Baylor University)—379.

*Inheritance of Susceptibility to Caries in Albino Rats (Mus Norvegicus). H. R. Hunt, C. A. Hoppert and W. G. Erwin (Michigan State College and Henderson State Teachers College)—385.

Effect of Temperature on the Rate of Setting of Plaster of Paris

HOWARD K. WORNER

THE setting time of six dental plasters has been determined at temperatures ranging between 10 and 100 C. There is an appreciable decrease in setting time as the temperature of the environment is raised from 10 C. to about 30 C. Over the range from 30 to 50 C., the setting rate is practically constant and is a maximum for the whole temperature range. At temperatures above 50 to 60 C., the setting times begin to increase again, and, with slow setting plasters, the retardation becomes quite marked at 70 C. At temperatures above 80 C., all plasters become very slow setting,

and, at 90 C., there may be little or no evidence of setting in five to six hours. No setting occurs at 100 C.

E. W. SKINNER.

Nasomeatal Line as a Guide for the Determination of the Occlusal Plane

WENDELL L. WYLIE

IT has been assumed that a line from the tragus of the ear to the ala of the nose is parallel to the normal occlusal plane in adults. This guide line has been used in compete denture prosthesis. In this study, the validity of the assumption was tested by measuring, in children and in adults with their own teeth, the angular relationship between the guide line and the occlusal plane. Measurements were made on tracings from Broadbent cephalometric films. In adults with a mean age of 40.5 years, the lines tended to converge anteriorly by 4 degrees. In children with a mean age of 10 years, the lines were parallel. The difference is statistically significant, and attributable to the porion's following a cranial growth pattern, while the other three points involved are facial points and reflect the movement of growth over a long period. The guide remains clinically useful.

Diabetes in Relation to Certain Oral and Systemic Problems. Part I

DANIEL E. ZISKIN, ELI H. SIEGEL and WINIFRED C. LOUGHLIN

DATA directed at ascertaining the relationship between diabetes and growth and development as a whole, and the teeth in particular, were analyzed by statistical methods in the cases of eighty-one white male and female juvenile diabetics. Patients, aged 4 to 19 years, were mainly from a camp of the New York Diabetes Association. Duration of the diabetes was from three months to fourteen years. Procedures were as follows: 1. Height percentiles, arrived at according to Burgess, were compared with her normal standards. 2. By means of the Klein, Palmer and Knutson DMF tooth caries experience grading, differences between their normals and this group were analyzed. 3. The number of permanent teeth erupted was compared with numbers in published reports on school children. 4. Roentgenographic data on root development were compared with McCall and Schour standards. Oral and physical examinations were supplemented by histories and previous records detailing sexual maturity, insulin dosage, diet, diabetic control and nutritional status, especially deficiencies. All statistical analyses were insignificant. Contrary to previously published reports, these data indicate that precocious general, sex and dental development cannot be linked *per se* to the diabetes. Reports that some factors in diabetic control reduce the incidence of tooth decay were not confirmed. Our results minimize the theory of control of dental caries by nutritional means via general metabolism. A striking finding in 92 per cent of the cases was the marked change in appearance of the gingivae. They were violaceous, thickened and of increased crevicular depth. These changes were associated with hyperplasia, hyperkeratinization and increased glycogen deposits in the epithelium.

Reestablishment of Transseptal Fibers Following Extraction

S. W. CHASE and JOHN REVESZ

EXTRACTION of a tooth self-evidently results in extensive destruction of the transseptal fibers between this tooth and the approximating teeth. When deciduous second molars of macaques were extracted, new transseptal fibers were formed, joining the first deciduous with the first permanent molar, and bridging the extraction space. The reorganized fiber group consists of a dense band of nearly parallel collagenous fibers closely resembling the ligament-like normal transseptal fibers. Similar reorganized transseptal fibers are found also bridging the extraction spaces of human jaws. In the macaque, the reorganization is completed in as little time as five weeks and takes place in spite of the force exerted by an orthodontic appliance designed to produce continuous traction on the teeth joined by the new tissue.

Pain Threshold Raising Effects of Amphetamine

D. Y. BURRILL, F. R. GOETZL, and A. C. IVY

THE pain threshold raising effects of administration of various drugs were tested in human beings by means of an electric current

applied to teeth through fillings. The figures obtained were in terms of voltages, which were interpreted as measurements of the pain threshold. It was found that the isomeres of amphetamine are relatively potent analgesics as tested by this method. The threshold raising action of levo-amphetamine diminishes remarkably on repeated administration. This is not true of dextro-amphetamine. Neither dextro-amphetamine nor levo-amphetamine has any influence on the effect of later administration of the isomere. No explanation of these results is available.

Experimental Papillectomy Part II: Histological Study (Preliminary Report)

WILLIAM LEFKOWITZ, C. F. BODECKER, and H. H. SHAPIRO

THE intact dental papilla was removed from the developing permanent mandibular canine teeth in two cats. Histologic examination showed that eruption of the teeth continued at a retarded rate. The papillary area was repaired with connective tissue and bone. The inner surface of the dentin was covered with a bonelike tissue resembling cementum. The connective tissue attaching the cementum-like lining to the bone was similar to peridental membrane. Clusters of epithelial cells in the area were encapsulated by collagenous fibers. Later, bone formation occurred in these areas. Growth of enamel and dentin was arrested. The fact that calcification of the enamel did not progress beyond the matrix stage demonstrates that calcification of the enamel is dependent on a normal pulp. The ameloblasts degenerated into squamous epithelium and formed small cysts.

Studies on the pH of Saliva

L. L. EISENBRANDT

THE glass electrode was used to determine the pH of unstimulated saliva within one minute after removal from the mouth. The experiment continued for one year, 1552 tests being made on seven subjects at 9 and 11 a.m. and 1, 3, and 5 p.m. for one week each month. The data were studied statistically.

The mean pH was 6.64, a lower average than usually reported. The pH for the group varied markedly during the year and strongly suggested seasonal variations. Subjects varied from one another in annual and monthly means. Each subject tended to follow the trend of the group although displaying individualistic pH characteristics. The pH of saliva for the group was lowest at 9 a.m. (6.55), passed through the annual mean just after lunch at 1 p.m. and was highest at 5 p.m. (6.71).

In generalization, each subject has a mean pH of saliva that is characteristic for him. He deviates from this mean because of seasonal, monthly, diurnal and individual conditions. These deviations are similar in pattern to those of other subjects, but vary in magnitude. This study indicates that 1 p.m. is the best time to determine the pH of saliva when based upon single readings. In all data, the date and time of day should be recorded.

Mutations of Bacteria Under Dietary Influence

P. H. and L. J. BELDING

THE geographic distribution of the degenerative diseases of civilization parallels in an exact and striking manner the world-wide distribution of those diseases of mankind and animals which are known or are alleged to be caused by streptococci.

Primitive people, regardless of their geographic distribution, have in common two factors of great fundamental importance: a natural unmanipulated diet and a non-pathogenic basal body flora. The basal body flora of isolated communities living in the tropics, in the temperate zone and in the arctic regions is remarkably constant. It is characterized primarily by a lack of pathogenic streptococci, pneumococci, diphtheria bacilli and other pathogenic organisms. The significant difference, bacteriologically, between primitive people, domesticated and wild animals and breast-fed babies, on the one hand, and civilized human adults on the other, is the constant presence of pathogenic streptococci in the basal flora of the latter group and the absence of pathogenic streptococci in the former group.

The characteristic and striking difference in the flora of the saliva from breast-fed babies and the majority of civilized adults, when streaked on sucrose-containing medium and incubated forty-eight hours, is the absence of the mucoid streptococcal colony from the plate of the former, and its presence

on the plate of the latter. It is this mucoid form of *S. salivarius* that has been named the odontolytic variant or, more specifically, *Streptococcus odontolyticus.*

Within a short time after a baby is taken from the breast and placed on a diet containing sucrose and cereal, changes occur in the oral flora in a direction leading toward the oral flora that is found in adult civilized human beings. The flora becomes more complex and more acidogenic, pathogens occur with frequency and many of the streptococci exist in a transitional or frankly mucoid state.

Once the "shift" to the acidogenic state has taken place in the formerly primitive flora and the oral streptococci have become converted from a smooth vegetative status to the rapidly growing mucoid or evolutionary phase, virtually all carbohydrates consumed by man can be converted into acid with great rapidity. Thus, after the condition of caries hypersusceptibility, as demonstrated by the presence of many organisms in the mucoid phase, has been established, *all* carbohydrates consumed by man, perhaps even protein, contribute to the carious process.

Inheritance of Susceptibility to Caries in Albino Rats (Mus Norvegicus)

H. R. HUNT, C. A. HOPPERT, and W. G. ERWIN

A GENETIC study of susceptibility to dental caries in rats was made, using the Hoppert, Webber and Caniff caries-producing diet. Phenotypic selection, progeny testing and brother by sister inbreeding have produced a susceptible and a resistant line. Eleven generations of the susceptible (1357 rats) and six generations of the resistant (1193 rats) are included in this report. Resistance was measured by days elapsing from introduction to the foregoing diet (at 35 days of age) to the appearance of the first cavities in the lower molars. Sizes of sibships might have influenced average performance unless this factor had been eliminated in computation. The mean resistance of each sibship was secured, then the average of these sibship means in a generation was computed. The average of sibship means in the seventh generation of the susceptible was thirty-eight days, of the resistants 245 days. The *t* value of the difference (207 days) was 7.31. The odds against this difference being due to chance exceed 2,000,000 to 1. Thus, with diet held constant, hereditary differences can be demonstrated in rats with respect to dental caries. The research is continuing.

Histopathology of Enamel Caries

BERNHARD GOTTLIEB

THE carious process first invades the prism sheaths while the prisms between appear to be intact. Later, the process invades the prisms and increases their resistance to acid. In the advanced stage, the prism sheaths become black in the decalcified section and finally disappear entirely, apparently being digested. The prisms remain visible, separated and no longer connected by the prism sheaths. Finally, they become necrotic too. Thus, four stages can be differentiated in the progress of enamel caries: (1) the organic frame work is thickened; (2) the process invades the prisms, making them more acid-resistant, while the prism sheaths turn black; (3) the prism sheaths disappear while the isolated prisms are still present, and (4) a necrotic mass replaces the enamel structure.

Penicillin—with Special Reference to Its Use in Infections Complicating Diabetes

THE efficacy of penicillin in the treatment of certain pyogenic infections, together with its low toxicity, makes the drug useful in the management of certain infections associated with diabetes mellitus. It is well adapted for use in the pyogenic infections, such as carbuncle. A report of fifteen cases suggests that it may be more effective if applied locally, directly on the infected tissue, than if it were injected parenterally for its systemic effect. No direct influence on the patient's insulin requirement has been noted. —*Am. J. M. Sc.*, 208:597, November 1944.

PAUL H. KEYES.

Fluorine and Dental Caries: Further Investigation

ROBERT WEAVER

A PREVIOUS report showed that the low caries incidence in South Shield (England) school children as compared with North Shield children might be due to the fluorine content of the South Shield water supply.

Further investigation suggests that fluorine is a caries-postponing rather than a caries-preventing factor. Examination of older pupils and of mothers in North and South Shield indicated that the average postponement probably does not exceed five years, but it may be greater in certain individuals. Examination of 800 children who had come in contact with fluorine at various ages during childhood suggests that fluorine protection is obtained either during a brief period before eruption or just after eruption. There is evidence that the influence is exerted on the teeth during the preeruptive period.—*Brit. D. J.*, 77:193, October 6, 1944.

PAUL H. KEYES.

Saliva Glucose: A Quantitative Method for Its Determination in Young Children

C. M. KORTUEM

THE Folin and Folin-Malmros micromethod for the quantitative determination of blood sugar was modified for the determination of salivary sugar in samples of 0.2 cc. size. The saliva of thirty-one healthy infants, ranging in age from 15 to 40 months, and living in an institutional environment was used for this study. After fourteen hours of fasting, the two-dose method of Exton-Rose was employed to establish a glucose tolerance curve for the blood and saliva. Twelve and one-half grams of dextrose in 80 cc. of 15 per cent lemon juice was given at the half-hour periods. The salivary curve rose from a mean of 10.6 mg. of glucose per hundred cubic centimeters of saliva, while fasting, to 51.8 mg. at the end of a 60-minute period. It fell to 21.3 mg. fifteen minutes later.—*Am. J. Clin. Path.*, 14:70 (Tech. Sect.), July 1944.

J. E. GILDA.

Dental Caries in the Cotton Rat: I. Methods of Study and Preliminary Nutritional Experiments

J. H. SHAW, B. S. SCHWEIGERT, J. M. MCINTIRE, C. A. ELVEHJEM and P. H. PHILLIPS

IN a report on the susceptibility to dental caries of the molar teeth of cotton rats raised from weaning on synthetic rations high in sucrose, describing the type of dentition present, the regions susceptible to caries and a method of recording the position and extent of the lesions, the authors state that almost no tooth decay occurred in those animals fed a stock ration composed of natural foodstuffs or a synthetic ration in which sucrose had been replaced by dextrin. An experimental period of fourteen weeks was found to be optimal for the observation and evaluation of the lesions. The rate of incidence or extent of the cavities appeared not to be affected by either the particle size of the rations or the addition of liver supplements to the sucrose rations. The liver supplements were given to produce optimum growth. Increase in the amounts of the fat-soluble vitamins, A, D, E and K caused a decrease in the number and the extent of the carious lesions in those rats on one of the basal rations. There was a definite bilateral distribution of the carious lesions. There was a much lower caries incidence in the molars of the upper jaw than in those of the lower jaw. The ascending order of caries incidence in the molars was as follows: upper first, lower first, upper third, upper second, lower second and lower third. —*J. Nutrition*, 28:333, September 1944.

J. E. GILDA.

Ulcerative Colitis Caused by Vincent's Organisms

A. HURST and R. L. VOLLUM

IN a case of ulcerative colitis in a man, aged 30, the onset of diarrhea was very gradual, as regards both frequency and liquefaction. The symptoms increased in severity until there were nine or ten blood-stained, slimy stools a day. There was no pain, no fever and no history of sore throat. Endoscopic examination revealed an atypical picture resembling that of amebic dysentery. The mucous membrane was covered with small ulcers, some round and some irregular in shape, all with thick, deeply congested, hemorrhagic borders. From the ulcers, large quantities of pus and some bright red blood exuded. Films made from a swab of the ulcers revealed large masses of fusiform bacilli and spirochetes of Vincent's type. The patient made a complete return to normal under neoarsphenamine therapy. This was attended by a concomitant reduction in number of the fusiform bacilli and spirochetes. —*Guy's Hosp. Rep.*, 22 (Fourth Series):118, 1943.

J. E. GILDA.

ANNOUNCEMENTS

CALENDAR OF MEETINGS

National

American Academy for Plastics Research in Dentistry, Chicago, February 11. Sylvester W. Cotter, 11059 S. Hale Ave., Chicago 43, Secretary.

American Academy of Periodontology (Council Meeting), Chicago, February 10. Raymond E. Johnson, Lowry Medical Arts Bldg., St. Paul 2, Secretary.

American Academy of Restorative Dentistry, Chicago, February 10-11. W. Samuel Tucker, 940 Canal Bldg., New Orleans 12, Secretary. (Canceled.)

American Association of Endodontists, Chicago, February 15-16. Louis I. Grossman, 4001 Spruce St., Philadelphia 4, Secretary.

American Association of Industrial Dentists, Chicago, April 23-28. Ernest Goldhorn, 11001 S. Cottage Grove Ave., Chicago 28, President.

American Association of Orthodontists, Colorado Springs, May 21-24. Max E. Ernst, 1250 Lowry Medical Arts Bldg., St. Paul 2, Secretary.

American Board of Orthodontics, Colorado Springs, May 17-20. Bernard G. deVries, 705 Medical Arts Bldg., Minneapolis, Secretary.

American Dietetic Association, Cincinnati, October 15-19.

American Society for the Advancement of General Anesthesia in Dentistry, New York, March 26. M. Hillel Feldman, 730 Fifth Ave., New York, Secretary.

Association of American Women Dentists, Chicago, February 11-12. Muriel K. G. Robinson, 4906 Walnut St., Philadelphia, Secretary.

State and Local

Chicago Dental Society Midwinter Meeting, February 12-15. H. A. Hartley, 30 N. Michigan Ave., Chicago 2, Secretary.

Cleveland Dental Society, May 7-9. Homer C. Alexander, 105 Windermere Theatre Bldg., East Cleveland, Program Chairman.

Cleveland Dental Society Children's Dental Health Day, February 5. C. F. Tuma, 13201 Miles Ave., Cleveland 5, Secretary.

Connecticut State Dental Association, Bridgeport, May 2-4. Earle S. Arnold, Secretary.

District of Columbia Dental Society, Washington, second Tuesday of each month. J. Walter Bernhard, 1835 Eye St., N. W., Washington, Secretary.

Hinman, Thomas P., Midwinter Clinic, Atlanta, March 25-27. S. A. Garrett, 302 Medical Arts Bldg., Atlanta, Chairman.

Illinois Alpha Omega, Chicago, February 11. Eugene S. Aron, 55 E. Washington St., Chicago, Secretary.

Illinois State Dental Society, Peoria, May 7-10. L. H. Jacob, 634 Jefferson Bldg., Peoria 2, Secretary.

Iowa State Dental Society, Des Moines, May 7-9. A. N. Humiston, 417 Higley Bldg., Cedar Rapids.

Iowa State Board of Dental Examiners, Iowa City, June 11-15. Harry G. Bolks, Sioux City, Secretary.

Kentucky State Dental Association, Louisville, April 2-4. J. L. Walker, 640 Barbee Way South, Louisville, Secretary.

Louisiana State Dental Society, New Orleans, April 24-26. F. Harold Wirth, 829 Maison Blanche Bldg., New Orleans 16, General Chairman.

Maine Dental Society, Portland, June 14-16. Alonzo H. Garcelon, Division of Dental Health, Augusta, Secretary.

Maryland State Dental Association, Baltimore, May 7-8. Paul A. Deems, 835 Park Ave, Baltimore 1, Secretary.

Massachusetts Dental Society, Boston, April 30-May 3. Evelyn F. Eaton, 106 Marlborough St., Boston, Executive Secretary.

Michigan State Dental Society, Detroit, April 16-18. S. G. Applegate, 630 E. Jefferson Ave., Detroit, General Chairman.

Minnesota State Dental Association, Minneapolis, February 27-March 1. L. M. Cruttenden, 498 Lowry Medical Arts Bldg., St. Paul 2, Secretary.

Mississippi Dental Association, Jackson, May 6-8. O. L. Colee, Magnolia, Secretary.

Missouri State Dental Society, St. Louis, May 14-16. C. W. Diggs, Exchange National Bank Bldg., Columbia.

New Jersey State Dental Society, Atlantic City, May 8-11. F. K. Heazelton, 223 E. Hanover St., Trenton, Secretary.

New York Society of Orthodontists, New York, March 5-6. Norman L. Hillyer, Professional Bldg., Hempstead, Secretary.

North Carolina State Board of Dental Examiners, Raleigh, June 25. Wilbert Jackson, Rich Bldg., Clinton, Secretary.

Oklahoma State Dental Society, Oklahoma City, April 15-18. E. W. Wise, Secretary.

Pennsylvania, Dental Council and Examining Board of the Commonwealth of, Philadelphia, April 16-21. Reuben E. V. Miller, Secretary.

Pennsylvania State Dental Society, Philadelphia, May 1-3. C. J. Hollister, 217 State St., Harrisburg, Executive Secretary.

Philadelphia, Greater, Annual Meeting, January 31-February 2. Abram Cohan, 269 S. 19th St., Philadelphia 3, Chairman.

South Carolina State Dental Association, Columbia, April 22-24. C. M. Douglas, State Hospital, Columbia, President.

South Dakota State Dental Society, Watertown, May 13-15. Ernest W. Elmen, Sioux Falls, Secretary.

Tennessee State Dental Association, Knoxville, May 14-17. E. Jeff Justis, 1504 Exchange Bldg., Memphis 3, Secretary.

Vermont State Dental Society, Burlington, May 23-24.

Virginia State Dental Association, Roanoke, April 23-25. J. E. John, Medical Arts Bldg., Roanoke, Secretary.

Washington State Dental Association, Spokane, June 21-23. G. D. Williams, 511 Paulsen Bldg., Spokane, President.

Wisconsin State Dental Society, March 19-21.

INTERNSHIPS, EXTERNSHIPS, RESIDENCIES AND FELLOWSHIPS

Bronx Hospital, Dental Department, New York. Director, S. Burton Gerstner, Fulton Ave. at 169th St., Bronx 56, N. Y.

Eastman Dental Dispensary, Rochester, N. Y. Director, Harvey J. Burkhart, P. O. Box 879, Rochester 3, N. Y.

Greater New York Conference of Hospital Dental Services. Secretary, Mary E. Reilly, 386 Fourth Ave., New York.

Post-Graduate Hospital, New York. Superintendent, W. B. Talbot, 303 E. Twentieth St., New York 3.

Research and Educational Hospital, University of Illinois Medical Center. Address Dean, University of Illinois, College of Dentistry, 808 S. Wood St., Chicago 12.

Walter G. Zoller Memorial Dental Clinic, University of Chicago. Address Director, Walter G. Zoller Memorial Dental Clinic, University of Chicago, Chicago 37.

Carnegie Fellowships for Research and Graduate Work in the Dental Sciences, University of Rochester. George H. Whipple, Rochester, Dean.

Research Fellowships in Medicine, Dentistry and Pharmacy, University of Illinois. Address Secretary of Committee on Graduate Work in Medicine, Dentistry and Pharmacy, 1853 W. Polk St., Chicago 12.